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Medicine

A Review of Therapy for Rheumatoid Arthritis

R. H. McFarlane, M.D.

A Review of Therapy for Rheumatoid Arthritis

Probably there is no condition other than rheumatoid arthritis for which so great a variety of therapeutic agents has at one time or another been advised. The very plethora of such therapies is of itself sufficient evidence that none is a cure, or anything near it. However, the past several decades have seen many useless methods discarded, and more recently, newer and more effective measures have become available. The advent of steroid therapy has in a dramatic way given evidence that the signs and symptoms of this disease are not necessarily irreversible; and these drugs therefore have been used widely, perhaps even indiscriminately, in the treatment of this disease. It seems worthwhile to review the progress of the past few years in an attempt to evaluate some of these measures, and to summarize present day usage. Such is the purpose of this publication.

The General Health of the Patient

Perhaps the first point to be considered is the attitude of the patient, and his doctor to the disease itself. There is almost always a great fear and anxiety in the mind of a patient who has, or thinks he may be developing this condition. It is first of all necessary to determine which of all these anxious enquirers actually has the disease; but the diagnosis having been made, the patient may need greatly some little assurance about the future. Chronic unrelieved anxiety is not good for any patient, and this holds true for the rheumatoid patient as well as others. Thus, it is well to recall that a substantial number of cases have spontaneous remissions of their disease, and that out of each 100 incipient instances, possibly some 75% may expect some kind of remission, more or less complete, and occurring either within a few months or less, or after a longer time. The patient may well be in the more fortunate group and the possibility of his becoming a completely helpless cripple is small, and severe crippling will almost certainly be long postponed, if it occurs at all. The attitude, "You have rheumatoid arthritis, and nothing can be done about it," has crippled many patients just as surely as the disease itself.

The next factor of importance has to do with such apparently simple matters as general hygiene, hours of rest, work, exposure to cold and fatigue. Naturally this is a highly individual matter; the balance between rest and exercise will be different for each person, and matters of work and economic necessity often must have a direct bearing on the kind of advice given. However, there are still those patients who need repeated cautioning against such apparently obvious things as overwork, fatigue and emotionally charged situations. Also, it must be clearly understood that rest for the whole body and the painful and vulnerable joints does not mean immobility with subsequent loss of joint function. It is equally important to be sure that the patient understands that exercise can be undertaken in ways that will not do injury to his joints. Many patients feel that they must "keep going" at all costs, and have produced permanent joint damage from overuse or trauma. Others, believing that rest is important, have allowed their joints to lie immobile until stiffened and perhaps deformed. It is of utmost value to be sure that so elementary a point is not overlooked. This may seem simple to the doctor; but often is not easily comprehended by the patient.

Some attention must be paid to the nutrition of the chronically ill in this as in other diseases, but apart from the provision of an adequate intake of protein, calories and possibly a suitable vitamin supplement, no dietary measures are of value. Overweight is not usually seen with rheumatoid disease, but when it is, it requires appropriate treatment.

The question of focal infection has come in for considerable attention in the last few years. In the past, possible foci of infection, such as teeth, tonsils, sinuses, prostate, and cervix uteri have been blamed for much in the etiology and progressiveness of the disease. At the present time it is generally agreed that infections in teeth, tonsils and other sites should be treated largely on their merits, and not with the expectation of altering the course of the arthritis. The removal of innocent teeth and tonsils is much to be deprecated; the more so since ill-advised surgery can be distinctly harmful. However, since there often is some discernible relationship between the severity of rheumatoid symptoms and exacerbations of certain extensive and frankly purulent chronic infections such as bronchiectasis and chronic sinusitis, these conditions do require careful attention when they exist. Cecil and Angevine[1] in 1938, reported on the frequency of foci in infection and the effectiveness of treatment of them in

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typical rheumatoid arthritis. In 200 cases they found relatively few foci, and the eradication of these proved beneficial in a very limited manner. This represents an almost complete reversal of the opinion of Cecil and Archer[2], who, a decade earlier, stated "successful treatment depends upon a relentless search for foci of infection and their prompt removal early in the course of the disease." The matter has been reviewed more recently by Lansbury[3] who felt that foci should be eradicated only if definite and obvious, and as a general health measure equally advisable in a person without rheumatoid arthritis. Ellman[4] has stressed the need for "a more conservative and discriminating attitude to the surgical attack on foci of infection." In 100 cases he felt that not more than 3% had genuinely benefitted from such treatment. Thus, current interest in the role of infection in what was once known as chronic infectious arthritis, is on the wane.

Many rheumatoid patients show a mild to moderate degree of anemia. Haemoglobin levels often run between 60 and 70%, and in only an occasional patient is the anemia severe, in the absence of some other factor. In most instances, it is a normocytic anemia with the mean corpuscular haemoglobin concentration but slightly reduced. However, many women have also an element of iron deficiency and this may be corrected simply enough. Apart from this, the anemia of rheumatoid arthritis is of a very stubborn nature, while the disease is still active. In cases treated with cortisone, the hemoglobin may rise temporarily while the steroid is administered, only to fall again when it is stopped. Ross[5] in 1950, studied the response to iron given intravenously. His second test series, consisting of 100 cases (50 treated with oral iron and 50 treated intravenously) showed, for the controls, a rise in haemoglobin from 71 to 79%, an increase of 8% only; and for the others, a rise from 69 to 84%, an increase of 15%. It took about three months for the full effect to occur. Sinclair and Duthie[6] used intravenous iron in 51 cases and noted a satisfactory response in 38 of them. After a follow-up of 7 to 23 months, the haemoglobin levels were maintained in 13 cases. In those who responded, the average haemoglobin at the start was 70%, after one month on oral iron, 71.8% and after one gram of intravenous iron, 84.3%. Curiously, in the seven cases so studied, no defect in the absorption of orally administered iron could be demonstrated. However, it is only fair to say that no confirmatory report exists in the American literature.

Transfusions have often been used to correct the anemia, but, as a rule, the effects are temporary only, and the transfused cells are destroyed in the body of the patient at the usual rate of red cell destruction. However, some have thought that lasting benefit might accrue, and that possibly remission in the course of the disease might follow transfusion therapy. Barsi[7] claimed exceptional results by transfusing the blood of pregnant women into rheumatoid patients, and concluded that the effect was due to some unknown substance circulating in the blood of these women.

The matter was recently summarized by Ebaugh[8] et al, who concluded that intravenous iron, Vitamin B12, folic acid and liver extracts have no place in the treatment of uncomplicated rheumatoid arthritis. They also concluded that transfusions were useless and even dangerous. Their only positive suggestion was that steroid therapy might be the most useful means of bringing the haemoglobin levels up, but that this too depended upon continued therapy.

The matter of heart disease deserves some mention. Whether its etiology be hypertensive, rheumatic or arteriosclerotic, its diagnosis is important. Quite apart from the diffculties imposed by heart disease itself, certain newer therapies for rheumatoid arthritis such as the steroids or phenylbutazone may be distinctly harmful if the heart is damaged. Thus the state of the heart may in some cases play an important part in the selection of treatment.

Relief of Pain

Since pain is so important a symptom, its relief is of some urgency. In many cases, adequate pain relief might be considered an end in itself, for it is sometimes evident that nothing else may be accomplished. In others, relief of pain will allow better joint function, even if nothing else is done.

It may seem redundant to mention the use of salicylates, and yet the writer is constantly surprised at the number of patients who have never been advised in the proper use of these drugs, which are inexpensive and safe for long-continued use. Acetyl-salicylic acid is surely a most effective pain-stopper for many individuals, and one that hardly ever produces any serious toxicity. Dosage may be pushed to tolerance, and three or four grams per day may be taken indefinitely. If tinnitus becomes unpleasant, simple reduction in dosage will usually correct it. Perhaps the chief deterrent to the use of aspirin is gastro-intestinal irritation, and occasionally this may be such that adequate dosage becomes impossible. Calcium acetyl-salicylate (Disprin) or salicylamide (Dolamide) may be better tolerated. Enteric coated tablets of sodium salicylate, or Pabalate, which has a very slowly soluble shell, may avoid this problem by breaking up in the intestine instead of in the stomach.

There are also many preparations available of salicylate in combination with other substances. Empiral (acetylsalicylic acid, phenacetin and phenobarbital) is inexpensive, and useful, and Phenaphen (phenacetin, acetylsalicylic acid, hyoscyamine sulphate and phenobarbital, with or without codeine) though more expensive is also useful, particularly

where some added sedative effect is desired. Para-amino-benzoic acid, and succinates have been said to enhance the effectiveness of salicylates but the actual evidence is difficult to evaluate. The Frosst formula of acetophen, phenacetin and caffeine, with or without codeine is universally used, though it often seems to the writer that in order to get enough salicylate, one may have to take an excessive dose of caffeine. By way of emphasizing the value of salicylate therapy, one might point to a recent study in Britain[9] in which results were indistinguishable in two series of cases — one treated with aspirin and the other with cortisone.

Codeine may be used in amounts deemed necessary. Demerol, in the hands of the writer, has not proved as useful as codeine. The use of morphine or any other habit-forming drug is mentioned only to be decried.

More recently a new anti-inflammatory and anti-rheumatic agent, phenylbutazone, has been introduced under the trade name Butazolidin. Used first in Europe, it has many similarities to aminopyrine. The latter drug is seldom used in North America due to its high rate of toxicity, although Butazolidin has been very readily accepted. A large number of publications have appeared dealing with the use of phenylbutazone in rheumatoid arthritis and other conditions. There is universal agreement that the drug provides relief from pain. Kuzell et al[10] noted that 69% of 117 cases had a good analgesic effect even when no other improvement occurred. Currie, Brown and Will[11] reported subjective improvement in 95%, improved performance in 93% (but objective evidence of improvement in only 30%) of 424 cases of rheumatoid disease. Frain and Morris[12] of Winnipeg stated that 72% of 40 cases had sufficient pain relief to warrant continued treatment.

Equally important, however, is a knowledge of the possible toxicity of this substance. Kuzell and his group noted 457 toxic reactions occurring in 40% of 800 patients given the drug. In 15% the medication had to be stopped. Stephens et al[13] noted toxic effects in 44% of 115 cases, and had to discontinue the medication in 17 cases, permanently in eleven. By contrast, Currie, Brown and Will recognized toxicity in only 4.7% of 424 cases, and only 3 patients were forced to discontinue medication. These authors characterized the drug as "remarkably free from serious toxic properties." Mauer[14] has recently summarized the findings in twenty-two cases of fatal phenylbutazone poisoning, and gives in detail the findings in a twenty-third fatal case, in which only 35 tablets, each 100 mgm were taken over a period of twenty-two days. The commoner causes of death were agranulocytosis (10 cases), peptic ulceration and gastro-intestinal bleeding (3 cases), exfoliative dermatitis (3 cases), and renal lesions (2 cases). Stephens noted thrombocytopenia in 16% of his cases (without purpura); but one fatality has occurred from thrombocytopenic purpura.

Among toxic effects, not necessarily fatal, gastro-intestinal symptoms, including acute peptic ulceration or reactivation of peptic ulcers, or bleeding from them, as well as less important symptoms were common. Skin rashes, agranulocytosis, or leucopenia, purpura and thrombocytopenia are frequent. Edema, sometimes with a rise of blood pressure, is so usual a complication that the writer considers it wisest to avoid using the drug where there is hypertension, any heart disease with or without congestive failure, and in elderly folks who show evidence of any appreciable amount of degenerative vascular disease.

It is also to be noted that toxicity can occur on doses of 300-400 mgm daily, but are much less likely to do so when doses are kept to this order than when larger. The writer has no desire to over-emphasize the dangers of phenylbutazone therapy, but wishes to express his opinion that a drug which is used only for relief of pain, should not be used until simpler and safer substances have been tried adequately and found wanting.

Measures Aimed at Altering the Course of the Disease

1. Vaccine Therapy

At the present time little interest is evidenced by rheumatologists in the use of vaccines. This has occurred because of the conviction that the disease is not of bacterial origin, and for the same reasons that focal infections are no longer considered of great importance.

2. Vitamin D

At the present time, interest in the use of high doses of Vitamin D has declined. By the late 1940's few rheumatologists considered it of real value, and in addition, it became evident that it was capable of producing serious toxic effects. Hypervitaminosis D is now a well enough known entity that many standard text books of medicine carry a few paragraphs on the subject. The use of massive doses of Vitamin D has therefore lost its place in current therapy.

3. Chrysotherapy

The use of gold salts for rheumatoid arthritis came into prominence after the reports of Forestier in 1929. Since that time, much has been written about the use of gold salts, but even now there is not unanimity of opinion amongst rheumatologists as to its value. Interestingly enough, its introduction into clinical therapeutics depended upon the misconception that many cases of rheumatoid arthritis were due to tuberculosis, for it had been previously shown that tubercle bacilli could be inhibited in vitro by gold cyanide. At present, those who advocate the use of gold must admit that its use is wholly empirical, and not based on information regarding a possible mode of action.

By 1937, Hartfall, Garland and Goldie[15] had reported on 750 cases, noting apparent cure in 9.9%, marked improvement in 56.8%, and moderate improvement in 13.0%. Weekly dosage was 100 mgm.

given intramuscularly. They had also discovered that gold salts were capable of producing serious toxicity and quoted 35.3% of their cases as showing reactions that were of some severity.

During the next decade a large number of papers were published on the subject of gold therapy. One could, by careful selection, find plausible publications which he might use in support of any opinion he might care to express. However, the majority of investigators found evidence that gold was of some value in some patients, particularly in early cases. Cecil et al[16] in 1942 reported 62% of 245 cases in remission or markedly improved from gold therapy, with the better results seen in cases of less than a year's duration. However, 42% of those benefited later had relapses. Again, in reviewing the problem of dosage, he concluded[17] that 50 mgm. of gold salt (Myochrysin or Solganol-B) given weekly, was the optimum amount, after a try-out on smaller starting doses given in the hope of detecting cases that might react badly to the treatment. His conclusions regarding total dosage were incomplete, but he felt that there was no point in going above a total of 1.0 Gm, in a single course, and that the total for each case might best be gauged by the response.

In 1946, Short, Beckman and Bauer[18] published a critical review of the literature up to that date, and presented their own experience at the Massachusetts General Hospital (limited to 47 cases treated with gold). These were compared to 274 cases handled similarly, except for the use of gold. In the gold series 60% improved as compared to 52.9% of the untreated series, and the authors did not think this was a significant difference. The figures for the control series also indicated that spontaneous remission occurred in 16% of the total, but none occurred in the gold-treated group. Their opinion was that the burden of proof still rested upon the advocates of chrysotherapy, and that "with the case as yet unproved for its place as a palliative or adjunct remedy in rheumatoid arthritis, the hazard of this form of therapy should furnish the decisive argument against its general use." Much the same evidence from the literature was reviewed by Hench[19] in 1947, but with dissimilar conclusions. He felt that "the use of gold salts seems entirely justified (1) in cases of progressive rheumatoid arthritis unrelieved by a reasonable, but not too long period of older and safer methods of treatment, (2) when the patient clearly understands and accepts the risk, and (3) when the physician is in a position to give the treatments with the necessary clinical and laboratory safeguards." With regard to the risk of toxicity, he points out that surgeons and patients alike will accept the risks involved in major operations to relieve symptoms more bearable than those of rheumatoid arthritis; and that operative mortality for major surgery in general is as great as that from chrysotherapy.

In the end, then, the decision as to the use of gold must be answered from each physician's own experience. The writer still holds the belief that gold in relatively early cases may prove of some value, and may be tried, provided the possible toxicity of the drug is recognized. The choice of preparations for practical purposes is limited to gold sodium thiomalate (Myochrysine), or gold thioglucose (Solganol-B), or gold thioglucoanilide (Lauron). The first two contain 50% of metallic gold and the other 54%. All are used by intramuscular injection. There is no need of using any gold preparation intravenously. Early workers used much larger doses than those new advocated, the smaller doses causing fewer toxic reactions. The excretion of gold is slow and the substance builds up in the blood and tissues. The studies of Freyberg[20] seem to indicate that a 50 mgm. weekly dose (25 mgm. of metallic gold) as the optimum one. This was also the opinion of Cecil[17]. The writer prefers to use a 10 mgm. test dose and if no ill effect occurs, carries on with a 25 mgm. weekly dose, and only occasionally a 50 mgm. weekly schedule. In this way only minor toxicity has been encountered. The total dosage is varied according to the progress made. If no effect is seen after 500 mgm. has been given, it seems unlikely that benefit will occur. If improvement occurs without the interference of toxic signs, the total is carried on to one gram. It is not usually necessary to go above this in any one course.

Toxic effects include skin lesions (anything from a minor erythema to an exfoliative dermatitis which may prove fatal), stomatitis, gastrointestinal symptoms (varying from slight nausea and anorexia to severe ulcerative enterocolitis) and nephritis. Hematologic disorders are more common, including leucopenia, agranulocytosis, hypoplastic or aplastic anaemia, and thrombocytopenic purpura. Some fatalities have been ascribed to each of these lesions.

Toxicity from gold is perhaps a less fearsome occurrence now than previously, since it has been shown that B.A.L. (British Anti-Lewisite) can act as an effective antidote, apparently "competing" with the tissues for the gold, and uniting with it to form an inert and stable compound. Because B.A.L. is not entirely innocuous itself, it need not be given for minor intoxications which may be corrected by the simple withdrawal of the gold. Particularly interesting in this regard is the report of Ragan and Boots[21] who treated five cases of gold dermatitis with B.A.L. The good results in four cases were sufficiently prompt to lead to the conclusion that the B.A.L. was indeed responsible. Also in four of the five cases, the arthritis, which had become quiescent, flared up again as the B.A.L. became effective. Excretion of gold in the urine had been speeded up during treatment. This

might be taken as further evidence of the original effectiveness of the gold.

However, in the past five years, interest in the use of gold has declined, because of the advent of steroid therapy. As the latter is more fully explored, it may be that gold will again assume a more important place in therapy.

4. Steroid Therapy

Since the preliminary report of Hench, Kendall, Slocumb, and Polley[22] in April, 1949, interest in the treatment of rheumatoid arthritis has centered mainly around the use of steroid therapy. There have been literally hundreds of publications on these substances in the interval, and needless to say it is not possible to make any complete review of this tremendous bulk of information. However, it may be simpler to try to trace the main gist of opinion on the use of steroids in the treatment of rheumatoid arthritis.

In their original publication, Hench et al recorded results of treatment of fourteen cases with cortisone and two with A.C.T.H., with esssentially similar clinical and biochemical results. The dramatic ameliorating effect of both substances occurred within a matter of a few days, along with improvement in the sedimentation rates; and equally important, the disease recurred on cessation of treatment, also within a few days.

By April, 1950, the same authors[23] were able to report much more detailed observations on twenty-one patients who had received cortisone and six who had received A.C.T.H. It was by that time evident that A.C.T.H. exerted its similar effects by stimulating the adrenal glands to greatly excessive corticosteroid production. The original results were extended and amplified. The beneficial effects consisted in relief of pain, relief of stiffness, diminution of swelling and increase of joint motion, decrease of joint tenderness, and increase of joint function, better muscular strength, greater sense of well-being, increased appetite, mental stimulation and even euphoria, a tendency to correction of anaemia, and a reduction in sedimentation rate, sometimes to normal. These benefits occurred rapidly and dramatically, often being evident in a matter of hours after the first doses were given. Cortisone was used intramuscularly in doses of 300 mgm. on the first day, and later with maintenance doses of 100 mgm. daily. The best group of patients were relieved of about 90-95% of their disability. Maximum subjective benefit was seen in about a week, but the full objective effect was not seen for about three weeks usually, and occasionally it took as long as three months to occur. On withdrawal, the symptoms tended to recur rapidly, often starting within four days. However, several cases had fairly prolonged (weeks to several months) partial or nearly complete remissions. Marked rebound relapses were put down to temporary suppression of adrenal cortical function. This seemed to last only a few days. Some comparisons between cortisone and A.C.T.H. could be made at this time. Thus the two substances could not be compared on a weight for-weight basis, but it seemed that 100 mgm. of A.C.T.H. produced the equivalent of about 200 mgm. of cortisone. Also, A.C.T.H., being in aqueous solution, was more rapidly absorbed and utilized, and this necessitated administration every 6 hours, whereas cortisone, as a suspension of crystals, could be given as infrequently as once in the twenty-four hours, making it much more convenient to use. It was also shown that the effects of A.C.T.H. could not be produced where there was originally a defect in adrenal cortical function; its mode of action being to stimulate the adrenal to produce its own hormones.

Some discussion of side effects was also possible at this time; these occurring usually as a function of dosage and length of time of administration. These were found to be due to physiological effects, but were excessive and undesirable, due to the dosages used. Salt and water retention with edema and rise in weight was noted, often only temporarily. Other effects were rounding of the facial contour ("moon face"), increase in the size of the pelvic girdle, and deposition of retrocervical fat ("buffalo hump"). Acne and hirsutism were noted, diminished libido and potentia, and irregular menstruation and amenorrhea. There were also C.N.S. effects; euphoria, increased mental activity, nervousness, insomnia, parasthesias, and fluctuations in mood. Some had beadache, fullness in the head, and dizziness. All of these were reversible on the withdrawal of the hormone causing them.

Similar results had been found by this time by a number of investigators, reporting small numbers of cases treated with cortisone or A.C.T.H., but, while these were confirmatory, none added a great deal of new information of a clinical nature. A later publication by the Mayo group[24] mentions as signs of hypercortisonism, edema, neurologic upsets, delayed wound healing, potassium loss with hypokalemia, negative nitrogen balance, alteration of sugar tolerance, hypertension, acne, hirsutism and menstrual upsets. They also reported as evidence of suppression of adrenal cortical activity persisting for some weeks after the withdrawal of cortisone, a reduction in the excretion of 17-ketosteroids, reduction in the eosinophil response to A.C.T.H., and transient weakness and fatiguability. However, the same beneficial effects were again emphasized.

Later in the same year, Polley and Mason[25] reported on trials of many other steroid substances, but only A.C.T.H., cortisone, and 17-hydroxycortisone (hydrocortisone) and some adrenal cortical extracts containing significant amounts of the latter, had any anti-rheumatic effect. Their

rather extensive bibliography indicated that many other investigators were in agreement with these findings..

Boland and Headley[26], also using cortisone intramuscularly, had found in a series of 42 cases that when initially large suppressive doses were used, smaller maintenance doses could maintain a satisfactory state of remission in 76% of their cases. The best results were in the milder cases, of course, but doses averaging 32-65 mgm. daily, or 100 mgm. thrice weekly were effective, and signs of hypercortisonism occurred in only 8.3% of the cases as compared to 33% of those receiving 100 mgm. daily. Since the Mayo Clinic group had found doses of 75 mgm. daily usually to be ineffective in severe rheumatoid, the differences of dosage seem to be referable mostly to differences in the intensity of the disease.

Hench delivered his Nobel Lecture in Stockholm in December, 1950, and it is published in the Annals of Internal Medicine in January, 1952[27]. By this time a myriad of publications had been made, and the status of cortisone had in many important clinical aspects been worked out. It had been found that the drug could be used orally as well as intramuscularly, and that, in fact, its absorption and utilization could be made more nearly to aproach the physiological state by frequent small dose administration. By this time, also, some of the side effects were better known, so that it had been recommended that these drugs be used with caution in cases of hypertensive cardio-vascular disease, diabetes mellitus, tuberculosis, old rheumatic carditis with decompensation, latent or frank psychoses, marked osteoporosis associated with senility or rheumatoid arthritis, peptic ulcers, glomerulonephritis, and possibly active syphilis. Dealing with one of the untoward effects of cortisone administration, Slocumb and Lundy[28] outlined the care of such patients coming for surgical procedures. It was emphasized that, due to inhibition or actual atrophy of the adrenal cortex, these cases might go into sudden, unexplained, and prolonged shock as a result of the defect on their own adrenal mechanism. Far from advocating reduced cortisone dosage, it was found to be imperative to expand it, using extra doses of intramuscular cortisone for 48 hours pre-operatively and for 2 or 3 days post-operatively.

Ward, Polley, Slocumb, and Hench in 1953[29] gave in some detail an account of their practice in the use of cortisone, and it appears that little has been added to the methods of use since that time. By then, the idea of large initial doses had been given up; orally-administered cortisone had displaced the intramuscular route, continuous treatment was preferred to interrupted courses; and the scheme of maintenance dosage was envisaged as the smallest amount that would give a reasonable, if not complete, control of the disease without production of hypercortisonism or with only minimal side effects. Thus, only the most severe cases were started on 100 mgm. daily, and the milder ones were started on as little as 30-50 mgm. In general it was felt that maintenance dosage should not exceed 25 mgm. for children, 37.5 mgm. for menopausal or post-menopausal women, 50 mgm. for other women, and 75 mgm. for men. Withdrawal of the drug had to be done slowly by gradually decreasing the daily doses at weekly intervals in order not to produce a period of rebound activity of the disease. Absolute contraindications were active tuberculosis and psychotic states, and less compelling contra-indications were cardio-vascular or renal diseases, tendency to thrombo-embolic phenomena, diabetes mellitus, osteoporosis, peptic ulcer, and convulsive disorders. The use of long term cortisone therapy was instituted in all cases of active disease where conservative therapy with rest, physiotherapy, and salicylates was ineffective after several months of trial. Gold was relegated to a lesser place in the therapeutic armament, being reserved for those in whom cortisone could not be used or maintained. This brings us up to date on the use of cortisone itself, except that presently many rheumatologists are more conservative in their decisions as to starting cortisone therapy. Ward et al quoted follow-up results in 46 cases after 8 to 24 months with great relief in 20%, marked relief in 35%, moderate relief in 41%, and mild in 4%, and felt that this was far better than any previous type of treatment in their experience. Freyberg, Traeger[30], et al had reported in December, 1951, on 44 patients treated for from 100 to 406 days, and found that good to excellent anti-rheumatic effect could be maintained. Only two cases discontinued treatment due to poor results. These authors also had found doses of 50-75 mgm. to be usually effective. They did note that in five cases, gradually increasing doses were necessary to maintain the same result. They were also disappointed at the high incidence of relapse on discontinuing the drug even after long periods of treatment. They also stressed the unpleasantness of the "cortisone withdrawal syndrome" and the emotional disturbance which occurred so regularly at the patient's finding himself just as ill at the end as at the beginning of a long and expensive course of treatment. This has been one of the major reasons for reticence in starting steroid therapy in the past; and it still is. These authors concluded that there was no evidence that the course of the arthritis was ultimately altered favorably by the prolonged use of cortisone; that cortisone was not suitable at that time for routine use; and that it must not be considered a treatment in itself, but only an adjunct. Thus, even this early in the history of steroid therapy, some doubt was cast upon its ability to

cause actual alteration in the disease process, although in general it was felt that cortisone could be a useful and effective form of assistance.

Copeman and others in 1952[31] reported on 20 rather badly disabled cases, treated over a period of months with cortisone, and were able to report that 17 had recovered enough to go back to work or household duties. One case only had been able to maintain improvement after stopping treatment. This seemed an enthusiastic and encouraging report, but later, in September, 1955, the Second Report by the Joint Committee of the Medical Research Council and Nuffield Foundation[9] was published, Dr. Copeman being among the authors. Opinion had undergone a profound change, for now the conclusion, after a two year study of 60 cases (30 treated with cortisone and 31 with aspirin) was "that there has been remarkably little to choose between cortisone and aspirin in the management of this group of patients." The two groups apparently had diverged but little and were more noteworthy for their equalities than for their differences. It might be remarked that the cases selected for this study were described as early rheumatoid arthritis, and in this group one might therefore expect that some would do well on the simpler methods of treatment. However, it does emphasize the changing of opinion as the long term efficacy of cortisone is studied. First there was great enthusiasm and then the opposite, but the truth may well lie somewhere in between; and one is not yet prepared to write off the usefulness of steroid therapy, any more than one was inclined to write off the usefulness of gold therapy after the adverse article of Short and Bauer[18] in 1946.

Holbrook[32] has recently published results of a four-year study on the use of A.C.T.H., cortisone, and phenylbutazone for rheumatoid arthritis. In a group of 35 cases treated with A.C.T.H. and a group of 36 treated with cortisone with the intention of producing the maximal suppressive effect compatible with safety, the results were not good. Of the A.C.T.H. cases, only 7 stayed on the treatment for 2 years, and of these only one still held grade 1 or grade 2 improvement. Of the cortisone group, only 10 continued for the 2 years, and of these only 2 still continued to show their maximal improvement. In both groups the failure rate at four years was 95%. In another group of 60 cases, older people with a more chronic form of the disease, cortisone was used in minimal dosage, and here the purpose was not to suppress the disease completely, but only to find a state of tolerance to it, while other rehabilitative measures were employed. Worthwhile improvement remained in 26 cases at the end of 4 years. In discussing the results Holbrook expressed the opinion that, without such treatment, some of the earlier cases might have been expected to have a spontaneous remission, and that none did so. He suggested that the lack of remissions might be due to cortisone or A.C.T.H. interfering with some of the normal processes of healing or repair. Thus, another keen student of rheumatology had to emphasize the failure of cortisone over the long haul, but his work seemed to indicate that the best use of steroid therapy might be in the more chronic cases in which the objectives of treatment were more limited.

The search for a steroid preparation which would have the beneficial effects of cortisone without its drawbacks led to the trial of other steroids. The first effective one to be investigated was hydrocortisone. Evidence had been available to suggest that this was the principal gluco-corticoid substance of the adrenal, partly on the basis that cases stimulated with A.C.T.H. showed a urinary excretion of hydrocortisone, but not of cortisone itself. Boland[33] compared the effectiveness of the two substances and found that, milligram for milligram, hydrocortisone was superior. In order to produce comparable results, a lower dosage could be used; the anti-rheumatic potency of hydrocortisone apparently being about 50% greater than that of cortisone. This author also believed that the undesirable side effects of steroid therapy were less with hydrocortisone in terms of dosage needed for effective control.

The same author again in 1955[34] reported on the prolonged use of hydrocortisone in 159 cases of rheumatoid arthritis, treated with the idea of producing maximal therapeutic effect with doses that could be tolerated. These cases were followed for 9 to 36 months. Sixteen percent had stopped the treatment, but toxic effects and poor control of the disease accounted for only one-third of these. Eight percent were in remission and had stopped because they were well. Of those showing limited improvement, 62% had not done well from the start, and 22% had shown an increasing refractoriness to the drug. 62% had side effects that precluded proper dosage. 72% of the total group had an adequate result by the end of 6 months, but only 59% at the end of two years. One-third of the cases showed clear-cut evidence of progression of the disease in spite of therapy. He listed as deficiencies in this mode of treatment: (1) intervention of hormonal side effects, (2) tendency to aggravate certain co-existing pathological conditions, (3) development of refractoriness in some cases, and (4) failure to prevent progression of the disease in at least one-third of the cases.

Thus, while hydrocortisone is equally as effecive as cortisone in about two-thirds of the dosage, it is in a general way subject to almost all of the shortcomings of cortisone itself. In terms of practical therapeutics its effects are not greatly superior.

More recently still, two new steroids with very potent anti-rheumatic effects have been synthe-

sized. The one, prednisone (first described under the name metacortandracin) is an analogue of cortisone. The other, prednisolone (first described under the name metacortandralone, is an analogue of hydrocortisone. Prednisone is marketed as "Meticorten" and "Deltra" and prednisolone as "Metacortelone," "Hy-Deltra," and "Delta-Cortef." Bunim and Bollet[35] reported the first cases treated with metacortandralone (prednisolone). Although the original work was done on this substance ease of production put prednisone on the market first, and only later did prednisolone become generally available. It was at once evident that both were potent anti-rheumatic agents, being about 3 to 4 times as effective as cortisone on a milligram for milligram basis. Other confirmatory reports are also available[36, 37]. There seems no doubt now that prednisone is more potent and effective than either cortisone or hydrocortisone. It also seems likely that the production of the same "toxic" or "side-effects" will be seen, though probably less frequently. This may not be true of its effect on peptic ulceration, however, since this complication has already been described[38]. It may be, also, since balance studies showed no tendency to potassium diureses or sodium retention, that this will not be a problem, thus allowing its use in patients, such as those with heart disease, who would otherwise be precluded from taking one of the earlier steroids. It has also been suggested that since hydrocortisone is more effective than cortisone, its analogue, prednisolone may be more efective than prednisone. The author has two cases, neither of whom was doing too well on prednisone, who improved, subjectively at least, by changing to prednisolone in identical dosage. He has had no opportunity to attempt the reverse.

It is necessary to mention very briefly the matter of halogenated analogues of hydrocortisone, several of which have received a therapeutic trial for rheumatoid arthritis. Boland[39] reported the use of 9-alpha-fluorohydrocortisone in 13 cases. It was a much more effective anti-rheumatic agent on a weight for weight basis (about ten times) than hydrocortisone. However, very minute amounts consistently led to the development of gross salt and water retention, and it is therefore not practical to use it.

A form of therapy combining the use of both gold and steroids deserves passing mention. Not too much information is available, but a recent publication by Bilka and Weil[40] reports on 41 cases so treated. All had 500 mgm. or more of gold salt and either cortisone or A.C.T.H. as well. These patients were observed for at least three months after the cessation of steroid therapy. The idea was that, if the steroids were to become ineffective on continued use or produced too many side effects and therefore had to be stopped, perhaps a gold salt begun at the same time and used concomitantly and acting to produce its effect slowly, might maintain the remission obtained by the steroids. Apparently the method, which the authors describe as practical, allowed rapid control of symptoms, but the prolonged remissions induced by gold were about as numerous as the authors would have expected had the gold been used alone. Gold toxicity occurred during therapy, but was most noteworthy in the first month after cessation of steroid therapy, suggesting a protective effect against it by the steroids. The theory of combined usage is at best a nebulous one, because no reliable estimate exists of the effect of gold alone.

One might also mention briefly the recent marketing of several preparations containing small amounts of prednisone or prednisolone, with salicylates. This apparently is said to be the upshot of certain work done by Spies[41] in Alabama on the potentiating effect of salicylates on these steroids. The reader might bear in mind that no such potentiating effect could be found by the Mayo group with regard to cortisone, although the matter was explored there some years ago.[25]. The actual doses used in these preparations are of the order of 0.5 to 0.75 mgm. of the steroid along with 300 mgm. (gr.v) of aspirin. It scarcely need be pointed out, except to those who follow the Reader's Digest[42], that effective dosage of either substance at the discretion of the physician, does not necessarily depend on their being in the same tablet.

Intra-Articular Injection of Steroids

Hydrocortisone is used for this purpose; cortisone having been shown to have little effect, and what effect there was, being of very brief duration. The effect of hydrocortisone acetate is much more profound and lasting. Hollander[43] reports on 8,693 intra-articular injections in 852 patients (not all with rheumatoid arthritis). In this disease, the method can be used where (a) only one or two joints are affected and no sytemic therapy is needed; (b) where systemic therapy of any type has resulted in good control of the disease, but which might be augmented by local treatment of particular joints; (c) where systemic steroid therapy is contra-indicated and palliation for particular joints is needed; and (d) as an adjunct to other measures being used to correct deformity of a joint. It is necessary to remember that such treatment is not an end in itself, and also that no systemic effect will occur as a result of absorption of the hydrocortisone. It is also necessary to be sure that the substance is placed in the joint space; failure to get an effect often being due to extra-articular deposition of the material. It is also wise to aspirate the excess fluid before the steroid is instilled. Usually a successful palliation is achieved temporarily, possibly only for several weeks, or even a month or more. A pronounced local anti-rheumatic effect is noted, with reduction in swelling, local heat, and pain. In Hollander's

series, only 2.3% had any adverse reaction, and this usually consisted in a mild increase in the inflammatory reaction around the joint for a few hours or a day or so, but even when this did occur and again subsided, the end result was better usually than the condition was before the injection. In any case, such a reaction was unlikely to occur on subsequent injections in the same patient, or in the same joint. It has been suggested that perhaps the local use of hydrocortisone in this way might cause localized demineralization of the adjacent bone ends, as it is known that systematically administered steroids will cause demineralization. The writer knows of no evidence that this is indeed the case.

Recently information on this type of therapy has been brought up to date by Hollander[44], who has utilized a number of steroids in the search for one more effective than hydrocortisone acetate. An ester of hydrocortisone, its tertiary butylacetate ((Hydrocortone TBA) was demonstrated to have a more protracted effectiveness, although not necessarily a more profound effect per dose. 9-alpha-fluorohydrocortisone was used also, and 5 mgm. doses seemed equal to 37.5 mgm. of hydrocortisone, but when used in knees, 50% of the patients developed edema of the leg after the injection.

The dosage of hydrocortisone (acetate or TBA) is usually 25 mgm. for a small joint or 50 mgm. for a larger one. The frequency of administration depends on the duration of effectiveness for the particular case. If too frequent injections are needed the treatment is impractical, but when properly utilized, it has a definite though limited value in the management of rheumatoid arthritis. The actual injection usually is not difficult, but a sterile technique is of the utmost importance.

Physiotherapy and Rehabilitative Measures

Physiotherapy, although not in any sense of the word curative, has a definite value in rheumatoid arthritis, partly because the application of some physical agencies may relieve a great deal of pain, but more importantly because certain physical methods may prevent or reduce deformities of the joints. While it is an advantage to have access to a good department of physical medicine such as exists in many modern hospitals, a great deal of useful work can be done without this by the conscientious physician, the patient and the family. The lack of the finest in the way of equipment need not preclude the use of many simple home remedies, and the use of common sense in the care of inflamed joints.

The use of heat, for example, is soothing to the inflamed joint and contiguous structures, and its application often eases pain and associated muscle spasm. This in itself may allow more use of an inflamed joint. Infra-red radiation can be had from a carbon arc lamp, which is easily available in most places, or the use of an electrical heating pad; hot water bottles or hot fomentations may be equally acceptable. An expensive diathermy machine is not by any means necessary, and in the more acute inflammations is probably even harmful. This means of applying a penetrating form of heat is more useful in the more chronic forms of joint disease in which there is little of the inflammatory element in evidence. With a little effort and ingenuity a good wax bath to apply heat to the hands may be constructed for use at home.

The use of rest and exercise is another matter in which simple instruction to the patient or his family may be of some considerable help. As a general rule, the more acute the inflammatory process in a given joint, the greater its need for rest; and conversely, the less acute or more chronic the inflammatory process, the greater the need for exercise. For example, a very acutely inflamed joint may need rest so greatly that it may have to be splinted to provide it. In such a joint it may be possible at first to produce only a few degrees of motion gently and with assistance, and this should be attempted daily. As the inflammation dies down, so does pain and muscle spasm, and after a few days it is often possible for the joint to be assisted gently through a much better range of motion. It may then be that as healing progresses, the patient may be able to take more and more active exercise for the joint, at first increasing only the number of attempts to regain the full range of motion, and later using graded resistance exercises to build up muscle power again. This, of course, may be a very prolonged process, and in instances of chronic rheumatoid disease, it may seem well nigh interminable. However, in many of such cases, where the disease stays in a state of partial activity, the mere fact of not allowing the joint to slide into more and more disuse is in itself good progress. Whatever exercises are done, they must be such as to maintain the joint in its best position, and utilize the muscles in such a way as to prevent or not to accentuate deformity. And exercises must never be of such a nature as to add extra trauma to the joint. Attempts at passive stretching of joints are harmful. Usually heat applied before the exercises will make the process easier, less painful and more beneficial. The matter of how much exercise and how often is an individual concern, but in general some pain production during exercise is permissible provided it is not excessive, and does not last more than a few moments after the drill is done. More prolonged pain is an indication that too much is being attempted. Then, within the limits of what can be tolerated, one progresses from simple innocuous exercises to more strenuous ones when improvement occurs.

It may be worthwhile to consider several

joints separately, for they are often involved in rheumatoid arthritis, and in such a way as to produce typical deformities. A little simple advice may help prevent these.

In the hand, rheumatoid disease usually produces a typical pattern of deformity. The proximal interphalangeal joints are swollen, stiffened, and usually become fixed in partial flexion, although occasionally they may become hyperextended. The metacarpo-phalangeal joints are also usually involved, and these become flexed, and the fingers deviated ulnarwards. The wrist becomes flexed. Exercises for the hand should avoid the trauma of twisting, wringing or shearing motions, or of forced flexion which tends to pull the fingers still farther ulnarwards. Exercises that keep the hand in flexion for long periods should be limited, and the use of the extensor muscles encouraged. It is sometimes necessary to build splints for part-time use to prevent ulnar deviation during rest.

The knee is another joint that deserves special mention. Flexion deformities are frequently seen and are almost all preventable. One factor in the production of these deformities is the tendency to hold the painful knee in partial flexion for the sake of comfort. The pillow under the knee is the surest way to end up with a flexion deformity. The doctor may feel that this is too obvious to mention, but the patient may not know it. All that is necessary is to tell him. Also, disuse causes atrophy of muscle, and in the case of the knee the one most obviously affected is the quadriceps. Exercises, therefore, are utilized to keep this muscle in the best possible condition. Not nearly as much attention needs to be paid to the flexor muscles, which are naturally more bulky and powerful. Again it should not need to be stressed that exercises such as walking, climbing, kneeling, squatting and so forth are out. Non-weight bearing motion at first will keep the joint mobile and extensible, and later, if done against resistances, can build up a good deal of muscle power without trauma to the joint.

One should also mention briefly, the matter of rehabilitation of the more severely crippled patient. Quite a number of these "derelicts" exist, in nursing or private homes, a burden to themselves and to those around them. Lowman[45] of New York has recently published a study of a two-year attempt to rehabilitate some of these patients. In a group of 18 of the most severe cripples, at the Goldwater Memorial Hospital, treated with a program of "total rehabilitation," 14 had left hospital, and of these 7 were totally self-sufficient in their personal care, and the other 7 showed an average of 26% increase in functional capacity. One had been placed in a job. Of 20 cases not so badly crippled, 15 are now totally self-sufficient, and the others partially so with an average improvement in functional capacity of 29%. Seven have found full time jobs. Not every patient is suitable for such a comprehensive plan of attack as this institution was able to provide; and as Lowman pointed out, psychological factors were not unimportant in deciding who might be likely to make the best use of the course of treatment. It is evident that they were dealing with some difficult cases, as 49 patients were excluded from the study as being insufficiently crippled for consideration.

The foregoing has not been an attempt to deal thoroughly with the use of physiotherapy and rehabilitative procedures. Obviously, to do justice to this subject is beyond the scope of this paper, and would require the assistance of a psychiatrist and an orthopedic surgeon.

Summary

From this admittedly incomplete review of therapies currently used for rheumatoid arthritis, it is seen at once that there is no one treatment that will be universally effective, but that good results may be had only where painstaking care is given the patient over the course of his disease. However, the following points may be of some help:

1. The patient needs an understanding of his disease, but without undue emphasis on the threat to his future.
2. Attention to his general health is important.
3. There are simple and safe analgesic and physiotherapeutic measures which are often effective, and these ought to be the first line of defense.
4. When the above have not been sufficient to produce satisfactory results, other therapies such as phenylbutazone, gold and the steroids may be utilized. Their toxic effects and contra-indications have been discussed, but no direct indications for their use are given; nor can they be. However, the following points may help in this regard:

(a) Phenylbutazone has excellent pain-relieving properties, and little else. If pain can not be controlled, a trial of phenylbutazone may be useful.

(b) Gold salts may apparently hasten the occurrence of remissions in some cases. Therefore, relatively early cases that appear to be susceptible of improvement are the most appropriate for this therapy.

(c) The final evaluation of the steroids can not be made. It is true that the ones of choice now are prednisone and prednisolone. It may be that their best use will be in the more chronic cases, where incomplete control of the disease can be accepted, and in conjunction with other means of treatment.

5. Even the most severely crippled patients can be helped by a comprehensive program of treatment and rehabilitation, and if so, surely similar programs applied earlier will prevent much severe crippling.

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Book Review

Talking To Patients, by Brian Bird. This little book, written for the general practitioner by a graduate of the University of Manitoba, who has been trained in psychiatry and psychoanalysis, represents a very important trend in medicine today. This trend is not receiving the notoriety of the ataraxic drugs, but may in the long run be more important to medicine than these self same drugs. It is a book that has entirely to do with words; what words to use under different circumstances and with different patients. It was not very long ago that even in psychiatric centres the importance of what is said between doctor and patient was sufficiently emphasized. This is one of the debts medicine owes to the psychoanalytical school of thought, emphasizing and verbalizing the subtle interactions between doctor and patient in an interview.

This book is a practical one, and deals with certain specific situations. How to talk to the dying patient, how to talk to children of certain ages, how to deal with the matter of fees, how and what to say to the adolescent concerned over masturbation. These and many other topics are discussed individually.

The book is small and can be read through very quickly. However it bears study and re-reading for its own sake and for the message it carries. It brings the lessons learned on the psychoanalyst's couch to anyone who wishes to take a few moments out to read it.

There is nothing of the psychoanalytical jargon in it. It is written in clear, everyday language, and to anyone who is not acquainted with this contribution of the psychoanalyst, the thought that this is in any way related to psychoanalytical thinking will not occur. It, of course, has nothing to do with psychoanalytical dogma, it merely represents the fruit of intensive study of a person's conscious and subconscious reactions. J. M.

Pulmonary Hamartoma*

J. A. MacDonell, M.D.

During 1904 Albrecht, in describing fibrous tumours of the liver and kidneys, coined the term "hamartoma," a word derived from two Greek roots meaning "to err" and "tumor." Albrecht's definition is quoted[3] as a "tumor-like malformation in which, in truth, one can demonstrate only an abnormal mixture of normal developmental components of the organ in which they occur, whether it be with regard to the quantity, the arrangement, or to the degree of development, or in all three respects." When these tumors occur in the lungs, they are usually named according to the predominating tissue type, and thus may be chondromatous hamartomas or vascular hamartomas (hemangioma, telangiectasia, congenital arteriovenous shunts)[6, 7].

Of the benign lung tumors, the chondromatous hamartomas, with an autopsy incidence of 0.25%,[8] are second in frequency of occurrence to bronchial adenomas. The lesion is usually discovered between the third and the sixth decades. However, it has been reported in premature[3] and new born[2] infants and in 60 year old adults[4]. The tumor is twice as frequent amongst males as amongst females[4, 8]. The lesions are usually rounded, circumscribed, and subpleural in location[1, 4, 8, 10, 11], but may be endobronchial[1, 9], and occasionally diffuse[2]. Malignant change is a frequently mentioned possibility, but a rarely reported occurrence. If the hamartoma is located so as to involve major blood vessels and bronchi, mechanical compression may produce a clinically malignant course[5]. Grossly, the tumor is firm and slightly irregular in contour, the cut surface appears cartilaginous; microscopically, cartilage tissue predominates with clefts of bronchial epithelium and mucus filled spaces interspersed.

Pulmonary chondromatous hamartomas are usually discovered on routine chest X-rays, arousing the curiosity of the radiologist and the concern of the clinician. Although symptoms are seldom ascribed to their presence, the peripheral type may be associated with chest pain or discomfort and the endobronchial type with cough, hemoptysis or the manifestations of atelectasis. Definitive diagnosis requires endobronchial biopsy or thoracotomy, but calcification within a rounded lesion in a patient with no known previous tuberculous or mycotic pulmonary infections is strong presumptive diagnostic evidence[4].

The following case is illustrative of many of the usual features encountered in pulmonary chondromatous hamartomas.

Case Report

The patient enlisted in the R.C.A.F. during 1939 at the age of 25. His chest X-ray then was negative. While serving in England he developed a mild chronic cough. During 1945, because of a

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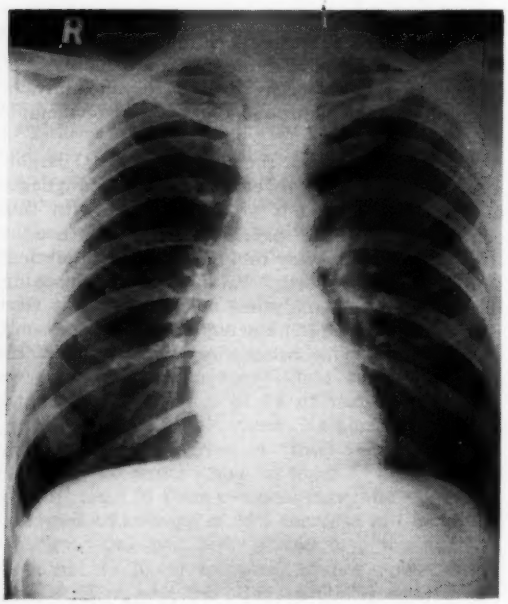
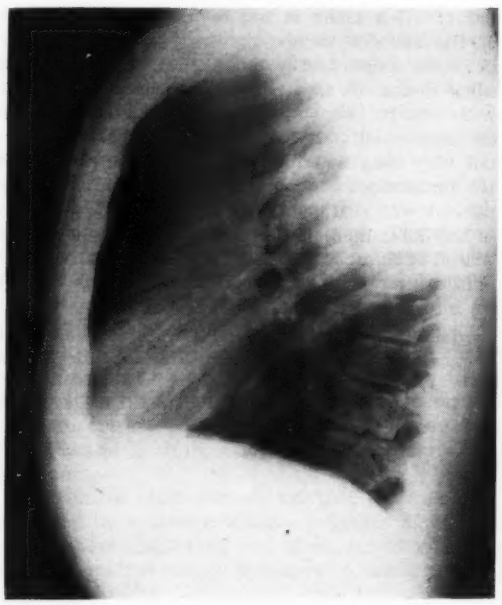
Figure 1 and Figure 2
P.A. and Lateral Chest X-rays 1945, showing circumscribed round lesion in lateral portion of the right lower lobe area.





cold, a chest X-ray was taken and revealed a small round shadow in the right lower lung field. Upon repatriation he was discharged from the services, but no definite diagnosis regarding his pulmonary lesion was made.

In 1949 he was admitted to Deer Lodge Hospital for investigation. He complained of a mild chronic cough and, during the previous three months, of the development of an aching right lower chest pain, more apparent when he was tired. Physical examination revealed no significant abnormalities, and a Casoni test, bronchoscopy and bronchography were likewise negative. However, radiologically, the lesion had doubled in size in comparison with the 1945 films and appeared as a homogeneous, circumscribed, round mass in the lateral portion of the right lower lobe (Figures 1 and 2). A thoracotomy was recommended; but the patient declined and was discharged from hospital to resume his occupation as a railroad switchman.

In 1955, having accepted the strong recommendation that the lesion be explored on the basis of further increase in its size radiologically (Figures 3 and 4), he was admitted to Deer Lodge Hospital. Again physical examination and routine laboratory tests revealed no significant abnormalities. The chest discomfort mentioned previously was still present and was unchanged. Irregular calcification in the central portion of the lesion was seen on tomography (Figure 5), and the radiologist suggested the diagnosis of hamartoma.

At thoracotomy, March 2, 1955, a large, firm tumor mass was found in the right lower lobe in a subpleural location. A simple incision of the tissue over the apex of the mass enabled it to be shelled out easily. Rapid section proved the lesion to be of a benign nature, and the chest was closed.

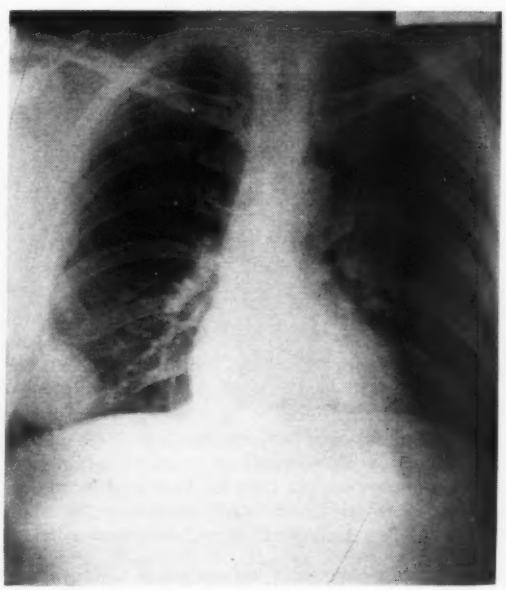
The tumor was firm, pale and globular. It measured 5.5 cms. in diameter and the cut surface was composed of several concentric areas, bluish in color resembling cartilaginous tissue. Microscopically, there were large areas of cartilage, numerous clefts lined by bronchial epithelium (Figure 6), some mucus and debris-filled spaces (Figure 7), and ordinary connective tissue.

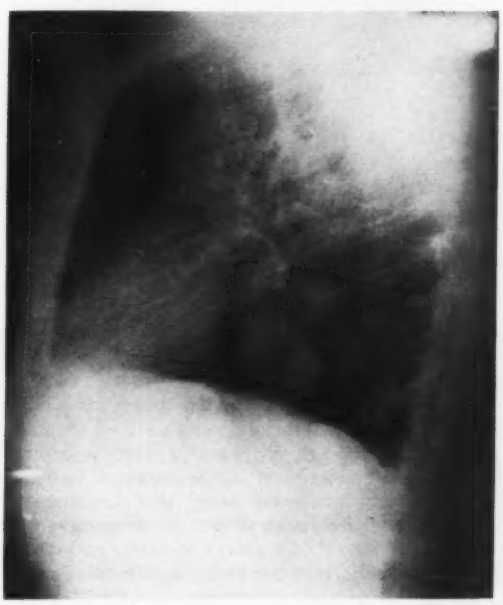
Post-operatively, subsequent to an episode of atelectasis necessitating bronchoscopic aspiration, the patient made an uneventful recovery and has remained well since.

Comment

The features of this case—an initially asymptomatic lesion discovered by a chance chest X-ray in a 31 year old male; a round, circumscribed lesion containing calcium, radiologically; a mass of firm consistency in a peripheral location at operation, which, on microscopic section, was composed predominantly of cartilage, but which also contained other connective tissue and bronchial

Figure 3 and Figure 4
P.A. and Lateral Chest X-rays 1955, illustrating an increase in size.





epithelium—are typical of the usual pulmonary hamartoma. Unusual are the length of time of observation prior to thoracotomy and the observed degree of growth[10] from no apparent radiological lesion on enlistment in 1939 to a 5.5 cms. mass in 1955.

Summary

The clinical, pathological and diagnostic features of pulmonary chondromatous hamartomas have been presented with an illustrative case report.

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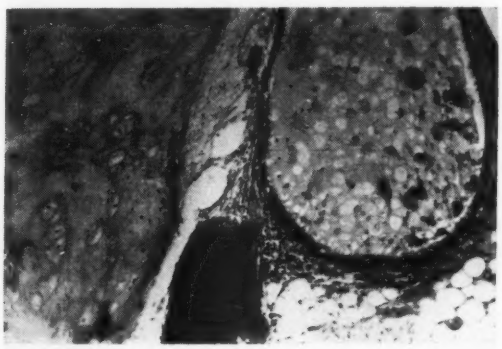
Figure 5
Tomograph of lesion, 1955, revealing clear cut outline and irregular calcification.

Figure 6
H and E stain (Low power). Illustrating large areas of cartilage and epithelium lined spaces.

Figure 7
H. and E stain (High Power). Areas of cartilage on left; mucous and debris filled, epithelium lined space on the right.



Obstetrics

Toxaemias of Pregnancy*

Elinor F. E. Black, M.D.

Toxic conditions arising from pregnancy are hyperemesis gravidarum, acute yellow atrophy of the liver and the pre-eclamptic and eclamptic state. It is with the latter toxaemia that I shall deal. We have effective treatment against two former killers in maternity patients, hemorrhage and infection, but pre-eclampsia and eclampsia continue to take a toll. It is only by constant awareness of the potential lethal significance of the triad of signs — abnormal weight gain, elevation of blood pressure, and proteinuria — in our pregnant patients that we shall be able to prevent and reduce the maternal mortality which is caused by toxaemia.

The cause of pre-eclampsia-eclampsia remains obscure, however constant research by many investigators is narrowing the field. One definite finding is that the highest incidence occurs in hot, humid climates, but this climatic factor does not prevent our patients in this northern latitude developing the condition. Also the incidence is higher where civilization has cast its supposed beneficence. Diet, environment, heredity, physical characteristics, psychological factors, allergies and chronic intoxications have no important causal significance. We know that it is a disease of pregnancy and predominantly of primigravidas; these two factors point the way to possible causes for our consideration.

First let us consider the kidneys. In the normal pregnant state it is known that the physiological functioning of the kidneys is under strain, but they manage to maintain the necessary excretory equilibrium in the majority of cases. However, any adverse conditions may turn physiological function into a pathological state with impairment of the proper elimination of water, sodium and chlorides, leading to electrolytic imbalance and oedema. This is the first break in the chain of normality, and hypertension and proteinuria quickly follow the original breakdown. Increased abdominal pressure from any cause will give decreased efficiency in kidney function which will eventually lead to oliguria and anuria; the increased abdominal pressure also causes hypofunction of the adrenal cortex with a resultant imbalance of sodium chloride metabolism. In pregnancy there is a constant increase in the intra-abdominal pressure as the months go by; as long as the kidney and adrenal cortex respond to this in a physiological manner all is well, but the balance between the physiological and the pathological appears to be a delicate one which can be upset in the later months of pregnancy. This theory of increased intra-abdominal pressure as a fundamental factor in the development of pre-eclampsia-eclampsia explains very satisfactorily the high incidence of the disease in primigravidas with their tense abdominal muscles; in multiple pregnancy and hydramnios where the uterus is over-distended, and in diabetes where both hydramnios and large babies are the rule. It also accounts for the normal pregnancies which follow a toxaemic one as it is seldom that the abdominal muscles regain their nulliparous tone.

Since the enlarging uterus is the cause of the increasing intra-abdominal pressure, we must look at it for possible intrinsic causes that might produce toxaemia. We know that pre-eclampsia-eclampsia is a disease of pregnancy only, and that it does not occur in women whose abdomens enlarge from the growth of ovarian neoplasms or fibroids. The most likely villain in this piece is the placenta as we know that the baby may suffer from the effects of toxaemia to the same lethal degree as its mother, although with different manifestations. The important factor about the placenta is its blood supply; if this is inadequate the placenta ceases to be a normally functioning organ. Placentation appears to be all important and if it occurs in a relatively ischaemic area of the uterus, a cornua or the lower segment, its blood supply may prove inadequate. If the decidua is poor, adequate placentation is difficult, threats to abort the embryo may occur, infarcts develop at the site of separation, and the maternal intervillous circulation is impeded. This same mechanism is operative in hydatidiform mole in which the oedema of the villi eliminates the intervillous channels. In the last trimester of pregnancy endarteritis of the arterioles and thrombosis of the capillaries commence. One of the essential lesions of the pre-eclamptic-eclamptic placenta is premature aging of the villi. The other is the production of fresh infarcts from recent premature separation; these are still sufficiently congested and thrombosed to interfere markedly with adequate circulation of the maternal blood. The rise in blood pressure which occurs at this stage may be of a compensatory nature in an endeavor to supply the needs of the foetus through what remains of healthy placental attachment. Whatever factors operate to separate the villi from their decidual attachment, it is obvious that lack of nourishment will cause degeneration of the villi and regression of the decidua with the possible production of tissue toxins which enter the maternal circulation and effect the liver and the kidneys.

That toxins are present in the circulating blood,

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which the liver attempts to detoxify to its own destruction, is shown by autopsy specimens. It is only in pre-eclampsia-eclampsia that the characteristic periportal hemorrhages and necrosis of the liver lobules occur. Also very striking in the gross specimens are the subcapsular hemorrhages, most frequent in the right lobe. It is postulated that the occurrence of a subcapsular hemorrhage in the eclamptic patient is the cause of the sudden epigastric pain of which the patients complain. The kidneys may show little change in the gross or there may be pallor of the cortex with congestion of the medulla. The glomeruli are oedematous and may be avascular. The main afferent arterioles show thickening of the endothelium almost to the point of occlusion. The collecting tubules are filled with protein and hemoglobin casts. These liver and kidney lesions are reversible as patients who have had severe eclampsia will recover and show no evidence of liver or kidney malfunction throughout the rest of their pregnancies or lives.

So much for theorizing on this interesting but baffling condition which is apt to crop up in our practice at any time. However, if one is alert there are definite signposts which warn us early if we have the eyes to see them. Let us consider some of the hints that are given us. First: an elevation of blood pressure found on the first early prenatal visit in a young patient. This may be due only to apprehension or excitement, but it is a warning that the patient has a labile blood pressure which will bear watching. The age of the patient of course will guide one's judgment in this matter, but, even if the elevation is mild in a woman over thirty, it is well to pay attention to it. If the elevation is extreme in the older patient one must decide whether she is a hypertensive, or a potential candidate for hypertensive disease, or if there is some renal disease either known to the patient or occult.

Second: if the patient is unduly obese, whether from fondness for food, hypothyroidism or heredity, she is a candidate for pre-eclampsia. Third: if the patient is of the fussy, neurotic, self-centred, apprehensive type she must be watched carefully. This type causes problems in differential assessment of symptoms, but one cannot afford to ignore her complaints even though one feels they are largely neurotic.

Fourth: if the patient shows signs of the pre-eclampsia triad before the 24th week of her pregnancy, one must consider the possibility of hydatidiform mole or chronic renal disease. This patient must be investigated immediately as to vascular-renal competency if there is no conclusive evidence of a molar pregnancy.

There should be no need for me to stress to this audience the necessity of adequate prenatal care with special attention to weight gain, blood pressure, and proteinuria as well as supervision of diet and elimination. These are fundamental essentials and any physician these days who does not follow a satisfactory routine in the care of pregnant patients should be labelled as professionally negligent. Every patient should be seen at least every two weeks after the 24th week of her pregnancy until the 36th week when she should be seen each week. Any untoward signs or symptoms mean that the patient must be seen more often than the usual routine. A group working in Australia have all but eliminated eclampsia by means of paying serious attention to the slightest deviation from normal standards in the pregnant patients and seeing them as often as once a week throughout pregnancy. I think that this is carrying things a bit too far. After all, pregnancy is a physiological condition, and I can see no reason for giving it a pathological connotation in the patient's mind by such frequent visits to her physician in the early months. Besides, I doubt if most of us have sufficient chairs in our waiting rooms to accommodate such assiduity in our prenatal care, barring the development of any untoward sign or symptom. I feel that one can be alert without being over-zealous. However, I urge that the first deviation from normal be taken seriously with regard to the patient's age, parity and previous history. Even dependent oedema in a multipara who is on her feet too much and has varicose veins to boot,warrants a little closer supervision although her blood pressure and urinalysis may be normal. Oedema of the lower extremities occurs in about 75% of cases during the last trimester of pregnancy and is probably due merely to back-pressure on the pelvic veins by the enlarging uterus. However, its appearance suddenly is no doubt the first indication that the delicate equilibrium of the kidney mechanism is being tipped from the physiological to the pathological. In a patient exhibiting such oedema it is wise to restrict the salt and baking soda in the diet, advise more rest and check her again in one week. If further weight gain has occurred on this regime and if the blood pressure has gone up from ten to twenty points, even though it still be below the arbitary level of 140/90, a grain to a grain and a half of barbiturate throughout the day together with bed rest should be prescribed. If at any time this type of patient shows albumin in a catheterized specimen of urine, hospitalization should be advised, unless oral doses of magnesium sulphate in sufficient quantity to produce two or three watery stools per day causes marked improvement in the toxic signs over a period of three days. In hospital the patient's diet, activities and elimination can be much more satisfactorily supervised and controlled than in the home, and it may be possible to release her in a few days to carry to term under watchful

supervision. I have found most patients quite cooperative in this regime, if the situation and its potential dangers to both the mother and her baby are explained.

I have described the early case with which one is apt to temporize in misplaced optimism. It is easier in the long run to be over-cautious with such prodromal signs and symptoms than to be faced later with the need for heroic measures to counter severe pre-eclampsia or eclampsia.

There is, of course, the case who has been pursuing a normal prenatal course under adequate care who presents herself showing the triad of pre-eclampsia. Such a case should be hospitalized immediately so that an anti-pre-eclamptic and sedative regime may be instituted together with control by daily urinalyses, twice daily blood pressure readings, and blood chemistry studies. These latter are important because they give one a guide as to the ensuing pattern of the physio-chemical balance. The eye grounds should be examined daily in these patients, as arterial spasm is readily seen and enables one to assess the degree of spasm throughout the rest of the body. It has been my experience that patients in this category of mild pre-eclampsia will show amelioration of signs for four or five days under treatment, remain stationary for another four or five days and then show a gradual increase in weight, blood pressure and proteinuria as the days go on. However, the time gained before they revert to their condition on admission to hospital may be sufficient to allow the foetus to achieve more maturity, as the end of term draws nearer.

This brings us to the question of the baby in the pre-eclamptic patient. Having postulated that the toxaemia is due to inadequacy of the placenta we must remember that the baby is growing under difficulties and is unlikely to be of normal size for the weeks of gestation; this is true even of the megafoetus of diabetes when a toxaemia is superimposed or where there is already vascular-renal disease due to the premature aging of the diabetic host. So we have a baby that may be called toxically premature. Added to this, it is not a good baby because it too has been affected by the electrolytic imbalance in the maternal circulation. There is no evidence to show that there is increased capillary fragility in the toxaemic mother, although there is increased capillary permeability. However, it is definitely known that the baby of a toxaemic mother has very definitely increased capillary fragility with a grave tendency to hemorrhages of the brain, lungs, adrenals, kidneys and gastro-intestinal tract. Thus the baby has three strikes against it even before it faces the hazards of labour and delivery.

The length of time for which a mild to severe pre-eclamptic patient should be carried along on treatment in the face of persisting signs has been arbitrarily set at three weeks. To temporize longer than this to bring her closer to term is thought to endanger the future of her vascular-renal system. I wonder if this prolongation of treatment has not lost babies that would have been safer in the hands of a pediatrician who is conversant with treating prematures than maintaining an inadequate growth and precarious existence in utero? Unfortunately one does not know at what moment the dire accident of abruptio placentae may occur, although this accident has been shown to be more apt to happen in the hypertensive patient than in the true pre-eclamptic one. Each case must be individualized as to response to treatment and severity of the condition, but obstetricians are now tending to terminate the pregnancy earlier in the course of pre-eclampsia, letting the baby take its chances with present methods of premature care rather than in the uncertain environment of the uterus.

Termination of the pregnancy in pre-eclamptic patients is not always easy. If the cervix is not taken up nor ripe, considerable difficulty may be encountered. In my opinion Caesarean section rarely has a place as a mode of delivery in pre-eclampsia either mild or severe. The patients are preponderantly young primigravidas and the chances of recurrent toxaemia in subsequent pregnancies are slight. Caesarean section is not as safe a method of delivery as per vaginam, and the baby still has three intrauterine strikes against it to militate adversely towards its ultimate survival.

When delivery is planned, I think all known measures for priming the uterus and ripening the cervix should be used, e.g., the administration of calcium and oestrogens in large amounts. Whether they are of any definite value is not known, but they do appear to have some efficacy in certain cases and they can be administered without harm to the patient in the days preceding more strenuous measures. Also, during these predelivery days, the mother should be given 10 mg. of Vitamin K parenterally twice daily; this is known to be of definite value in lessening hemorrhages in the newborn and may possibly be of some benefit to the mother.

The most efficacious way of inducing labour is by rupture of the membranes. If the patient is near term and the cervix is taken up, this is usually all that is necessary. If the baby is small and one fears prolapse of the cord due to cephalo-pelvic disproportion, the hind waters may be tapped with a Drew-Smythe catheter, providing one takes off a sufficient quantity of fluid, at least 16-20 ounces. If the cervix is not taken up, gentle dilatation to admit two fingers, stripping of the membranes from around the os, and rupture of the forewaters while the head is being pressed well into the lower uterine segment by a hand on the fundus is satisfactory, as the flow of fluid can be controlled while

the presenting part acts as a plug against prolapse of the cord. Labour usually ensues within 24 hours following release of the amniotic fluid, and very often there is amelioration of the toxic signs as a result of the decrease in the intrauterine pressure. If labour does not begin spontaneously within this time limit, I think that an intravenous pitocin drip, 5 units to 1000 cc. 5% glucose in water, run in at the rate of 20 drops per minute is the safest oxytocic we have. It is tiresome for the patient, but it gives the advantage of watching the blood pressure and oxytocic effect in the borderline patient. The rate of flow may be speeded up to 50 drops per minute if all factors appear to be under control. If the patient is unduly apprehensive, subcutaneous pitocin starting with ½ min. and repeating every ½ hour, increasing the dose to 1 min., then 2 min. for a total of three doses may be given.

Delivery should not be attempted until the cervix is fully dilated and the presenting part is well down. Prophylactic forceps are advisable to relieve the mother of the physical strain of second stage pains. Traumatic delivery must be avoided at all costs. Not only is it damaging to an already precarious baby, but also tissue trauma releases histamine into the maternal circulation and adds toxins to the already pathologically functioning liver and kidneys. Trauma to the maternal soft parts may throw an already sick patient into profound shock.

Despite one's efforts to give adequate prenatal care to all patients, cases of severe pre-eclampsia will occasionally occur. If a patient has a blood pressure above 160/110, persistent oedema over the tibias, and a proteinuria of .5%, she must be classed as a severe case on the borderline of eclampsia. In addition to the pre-eclampsia regime these patients must be given enough sedation to keep them mildly drowsy. The barbiturates will cause a decrease in blood pressure and may also cause a temporary oliguria, but they are effective in relieving headache and decreasing the patient's apprehension. The dose must be regulated according to the patient's tolerance and no arbitrary dosage can be laid down. Chloral hydrate is an excellent sedative for the toxaemic woman, but has not as marked an effect on the blood pressure as have the barbiturates. If the blood pressure rises to or above 200 systolic, intravenous magnesium sulphate, 20 cc. of a 10% solution or 10 cc. of a 20% solution may be used. This will cause a marked decrease in cerebral oedema with its attendant encephalopathy. It also causes a rapid fall in blood pressure which may make the patient nauseated, clammy and shaky. 12 cc. of a 50% solution injected intragluteally is equally effective and does not give such a sudden reaction; this can be repeated every six hours if necessary.

Again the eye grounds may be of use in assessing the progress of the disease. The severe eclamptic may show retinal haemorrhage and cotton wool patches.

Once the patient's condition has stabilized, steps must be taken to empty the uterus, for as long as the pregnancy is allowed to continue in these severe cases there is very real danger of eclampsia developing suddenly. Also there is a definite possibility of permanent damage being done to the vascular-renal system, as well as the probability of intrauterine death of the foetus or abruptio placentae. Induction of labour by rupture of the membranes is again the best way of terminating pregnancy. However, in a severe pre-eclamptic with an unripe cervix and in whom continuation of medical therapy while awaiting the onset of labour appears to be the path of folly. Caesarean section is probably the wisest choice in the interests of both mother and baby, providing the doctor is an experienced operator.

In eclampsia—and the severe pre-eclamptic who does not respond to treatment is to all intents and purposes an eclamptic who at any time may develop epigastric pain or convulsions — the most efective pre-convulsive treatment is intravenous glucose using 1000 ml. of a 20% solution, administered over 35 - 55 minutes. This infusion will cause cerebral dehydration, hemodilution, and diuresis without producing glycosuria. It can be repeated every 6 to 8 hours. If convulsions occur, routine steps must be taken for the care of the patient. These are: morphine gr. ¼ stat., turn the patient on the side, procure a gag, intranasal oxygen, pharyngeal suction, introduction of an indwelling catheter and application of the manometer cuff so that it can be used as a tourniquet for intravenous infusions as well as for frequent blood pressure readings. Intravenous or intragluteal magnesium sulphate should be administered and the intravenous 20% glucose solution started running. The catheter should be drained every hour in order that a close watch can be kept on the urinary excretion which should be in excess of 600 cc/24 hours, and the increase or decrease of proteinuria can also be estimated. Morphine gr. 1/6 may be repeated at hourly intervals until the convulsions are controlled unless the respirations decrease to 12 per minute; up to 1½ gr. per 24 hours may be given. Or sodium amytal 3¾ gr. subcutaneously may be given every 6-8 hours. Chloral hydrate or paraldehyde administered by rectum in suitable amounts are useful for prolonged effect.

No attempt to deliver the patient should be made until convulsions have been controlled for at least 24 hours. These patients are highly sensitive to any external stimuli and premature or injudicious attempts to terminate the pregnancy may produce a recurrence of the convulsive state. One is desirous of emptying the uterus, and in a

patient who has had convulsions the survival of the baby is fortuitous, therefore all measures are bent on saving the life of the mother. Very often labour commences during the treatment of the convulsive state and by the time the convulsions are controlled one is pleased to find the cervix taken up and already showing considerable dilatation. In these cases sedation of the patient must be maintained and delivery effected with as little trauma as possible. Cases in which convulsions cannot be controlled, and emptying of the uterus by Caesarean section becomes imperative to save the mother's life, are very rare.

One very real danger in eclampsia is cerebral haemorrhage. If a patient remains in coma following a convulsion this vascular accident must be considered as a possibility. Elevation of temperature and pulse rate are also unfavourable developments. It is because these untoward events can and do occur that one must keep conversant with the routine treatment of the eclamptic patient in order that early and effective treatment may be instituted immediately allowing no time for deterioration in the patient's condition.

If a patient has been a severe pre-eclamptic, it is advisable to give her morphine gr. ¼ following the third stage of labour. This sedation will prevent the development of post-partum eclampsia in many cases. The treatment of eclamptic convulsions in the early post-partum days is similar to that outlined for ante-partum or intra-partum eclampsia.

Articles are being written these days about the use of anti-hypertensive drugs, such as Apresoline, Rauwolfia and hexamethonium derivatives in the treatment of pre-eclampsia. It is true that blood pressure can be reduced by means of them and the risk of cerebral accidents decreased. However, they are operative in only one field of a highly complicated picture: the cause of the toxaemia is still present and the effects of it are still progressing. These drugs will reduce blood pressure and relieve arterial spasm, two effects which are highly desirable in the toxaemic host. But if the elevated blood pressure is one of Nature's intentional mechanisms to try to improve the placental circulation, our prolongation of the pregnancy by means of a hypotensive drug is just adding to the hazards for the baby.

Similarly mercurial diuretics and ammonium chloride will reduce oedema in the pre-eclamptic patient but have no beneficial effect on the underlying condition.

The use of cortisone and ACTH in toxaemia has proved to be of no benefit.

To summarize this brief review of a very complex subject, I would leave with you these points. We know that pre-eclampsia-eclampsia is due to pregnancy, and the disease cannot be completely overcome until the pregnancy is terminated. It is essential that we be ever alert for signs of incipient or impending toxaemia because we have responsibility for two patients, the mother and her babe. Once pre-eclampsia has developed we must take all possible measures to prevent it progressing to eclampsia. If eclampsia develops we must have ready knowledge of the effective routine of treatment in order to save the life of the mother even if we cannot always salvage the baby.

Oto-Laryngology

Vasomotor Rhinitis

I. H. Mazer, M.D., D.L.O. (Eng.)

Sufferers from this complaint remain one of the commonest and distressing problems of the rhinologist.

This condition is characterized by constant nasal stuffiness and increased post nasal discharge, accompanied by a dull headache and feeling of fatigue and nervousness. Often headache is the chief complaint, which causes the patient to seek medical help. In the past too, these cases have been improperly handled by the family physician, who advised the patient that he had a "touch of sinus" and suggested that he make the best of it. others have been given repeated vasoconstrictor nose drops with resultant abuse of the nasal mucosa due to secondary turgescence and swelling of the lining following temporary constriction.

Vasomotor rhinitis and allergic rhinitis are not synonymous. Allergic Rhinitis, as exemplified by hay fever, is characterized by vasodilation, edema, excessive secretion of mucus, and the presence of eosinophiles in the secretions and tissues. This antigen-antibody reaction causes a release of histamine, resulting in capillary dilatation, increased capillary permeability and tissue oedema.

Vasomotor rhinitis is not a disease of the nose, but a manifestation of a systemic disease, the result of which is a sympathetic-parasympathetic imbalance causing peripheral vasodilation, oedema and secretion. The common findings are a swelling of the turbinal mucosa with or without glairy mucus in the meatuses, a peculiar red or purplish color of the turbinates and excessive lymphoid nodules on the posterior pharyngeal wall and in the nasopharynx. Allergic testing and treatment do not offer any hopes of results.

The sympathetic nerves provide the vasomotor mechanism. Fibres are derived from the sphenopalatine ganglion. They enter in the Vidian nerve, having travelled in the great deep petrosal nerve from their origin on the plexus surrounding the internal carotid artery. Not only may there be physiological vascular alterations of wide degree, but also variations due to psychological influences.

In the mucosa of the turbinals are extensive vascular spaces, or erectile tissue, capable of distension by blood, which serve to warm and moisten the inspired air. Abnormal influences may upset the balance in the nose, resulting in swelling of the mucous membrane, or outpouring of an increased quantity of transudate.

Now let us consider the factors concerned in this vasomotor imbalance.

E. Endocrine Dysfunction

1. Thyroid Gland — Arthur Proetz and others have called our attention to vasomotor rhinitis occurring in the hypothyroid state. The symptoms occur here in conditions too minor to cause a general myxoedema in more rugged tissues. The nasal mucosa is red and dry, or often pale and boggy. The patient exhibits a slightly lowered B.M.R., high blood cholesterol and responds to thyroid extract.

A. R. Hollander prefers to call this state hypometabolism, in which there is a general slowing down of body functions without any obvious signs or positive tests of typical hypothyroidism. The new delicate test of blood protein-bound iodine has been used in place of B.M.R. Proetz showed that the response of the patient is more helpful than the changes in B.M.R. in determining the most effective dose of thyroid.

2. The Reproductive System—Nasal congestion is common at puberty before the menstrual period, and during the second half of pregnancy.

A recent case complained of a stuffy nose in the seventh month of pregnancy. The nose examination revealed pale, swollen turbinates. Two small polypi were present. When seen three weeks after parturition, the symptoms had disappeared, and the polypi were not visible.

Nasal blockage can occur in women due to menopause or castration. Oral and parenteral oestrogens will bring about cessation of symptoms.

In the male, vasomotor rhinitis can occur in the male climacterium. A male, age 60, had difficulty in breathing through the nose for about 3 years. He was advised by his physician to have his polyps removed. When examined, no polypi could be found. A careful endocrinal history revealed symptoms of male climacterium—weakness, nervousness, irritability and emotional instability. He received 10 mgm. testosterone propionate three times a week and later only once a week for one month. Afterwards he received one injection monthly. After two weeks, his nose breathing was free and he has had no difficulties since.

Very careful histories and endocrinological examination of cases with vasomotor rhinitis should be undertaken. It is possible to show definite relations between the different hormonal glands and the mucosa of the nose. The treatment is an attempt to restore the glands to normal function.

Watson-Williams of England reviewed a series of cases which revealed that spasmodic rhinorrhea and nasal congestion can occur immediately after coitus, equally in both sexes. In the more severe cases, the attacks lasted one or even two days, and presented all symptoms of a cold in the head, except fever.

B. Psychosomatic Factors

Emotional factors may play a part in chronic

organic changes in the nose. Wolff and co-workers have shown this to be true, and that acute emotional disturbances may cause eosinophilia in the tissues and in the peripheral blood. Allergy testing in these patients is usually negative. There is a definite relationship between stress and onset and course of vasomotor rhinitis. These cases will often refuse psychiatric treatment, and it becomes the task of the rhinologist to elicit the cause of the stress while giving symptomatic relief with sedation and vasoconstrictor drugs like ephedrine or propadrine.

In some cases, the precipitating causes may be a combination of psychosomatic and hormonal factors.

C. Diet

Patients with dietary indiscretions, taking a high carbohydrate diet, can develop vasomotor symptoms, in the nose. This is especially common in children.

Summary

An attempt has been made to show the necessity of careful clinical history and knowledge of the patient as a whole in evaluating his case.

Vasomotor Rhinitis is a manifestation of sympathetic-parasympathetic imbalance in which endocrine dysfunction plays an active part.

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Paediatrics

Idiopathic Spontaneous Hypoglycemia of Infancy

A Report of Three Cases and Discussion

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The hypoglycemia seen in young children is neither the same nor comparable to that seen in adults. In the few years since McQuarrie named and described the syndrome of "spontaneous idiopathic hypoglycemia of infancy," many cases have been reported from various centers. This is a condition which is found with more frequency in infants and young children than is diabetes mellitus.

The clinical picture is extremely varied and in no way pathognomonic. Some signs and symptoms are fatigue, weakness, flushing, sweating, speech and visual disturbances, incoordination, tremor, syncope, convulsions, and coma. It must be emphasized that none of these signs and symptoms is specific to this syndrome, and unless it is thought of, **it is likely to be missed.** Demonstration of the low blood sugar is necessary for the diagnosis.

Among the less common conditions which may produce hypoglycemia in children, one must consider disorders of the

	Tests
1. Pituitary	urinary ketosteroids
e.g., pituitary tumour	X-ray of skull (sella turcica)
2. Adrenal cortex	eosinophil depression test
e.g., Addison's disease	—adrenalin—ACTH
3. Thyroid	serum cholesterol
e.g. cretinism	protein bound iodide (P.B1)
4. Liver	liver function tests
e.g., von Gierke's disease	
5. Kidney	urinalysis (for glycosuria)
e.g., galactosuria	B.U.N.
6. Pancreas	insulin assay
e.g. tumour of islet cells	surgical exploration

Most of these conditions are evident on physical examination, even without the tests mentioned. An islet cell tumour of the pancreas may be very difficult to differentiate from idiopathic spontaneous hypoglycemia of infancy—in fact, it may be impossible to differentiate except by surgical exploration, but the incidence of such tumours in children is very low compared to adults. McQuarrie found only one islet cell tumour in a group of 40 hypoglycemic children, compared with 25 cases of idiopathic spontaneous hypoglycemia in the group. It is therefore felt that surgical exploration is not usually necessary in the case of an infant with hypoglycemia.

The need for early establishment of the diagnosis is very important because of the danger of permanent brain damage as a result of repeated hypoglycemic episodes. Any child with afebrile convulsions, coma, or "peculiar behaviour episodes" should have blood sugar estimations made —more than one may be necessary to demonstrate the hypoglycemia (cf. Case 1 below). The blood sugar specimen taken during or immediately after a convulsion may show a higher level of glucose

because of the response to physical activity, and should be repeated at a later time. The reported familial tendency of the syndrome was not demonstrable in our cases.

Case 1. V.H. Age 3 months.

This female child was the third born of a healthy mother, somewhat anemic; precipitate labor on the floor of a hotel room. Gestation 39 weeks; B.W. 5 lb. 4 oz. She did well for the first 5 weeks, when the mother noted that she lapsed into a semicomatose state from which she could be roused on vigorous shaking. These spells recurred 5-6 times in the next 24 hours. She did reasonably well in feeding and gaining weight, and had no more of these spells. However, the father stated that she was always much quieter than the other children had been, except when she was disturbed, and at these times she became very irritable. It was also noted that she perspired profusely.

At the age of 10 weeks the baby suddenly developed grand mal convulsions lasting 2-3 min., and recurring every 3-4 hours. Following a convulsion she was lethargic and hard to rouse. The family physician was unsuccessful in attempting to control the convulsions with sedation, and referred the patient to the Winnipeg Children's Hospital.

On admission the child was well-nourished but semicomatose (post-convulsive). The anterior fontanel was slightly full; otherwise, complete examination, including fundoscopic, was negative. The tentative diagnosis was subdural hematoma(s), possible birth injury, or cerebral agenesis. Investigations showed: skull films—neg.; serum Ca —4.64 mEq./1; BUN—18.8 mgm.%; blood sugar—71.4 mgm.%; spinal fluid — xanthochromic (previous trauma) with protein 66.5 mgm.% and 8 cells. Phenobarbitone and Dilantin failed to control the seizures. A ventriculogram showed slight dilatation of the ventricular system, no evidence of subdural affusions. An EEG was interpreted as normal. The child's clinical state was unchanged, with occasional convulsions followed by extreme lethargy.

On the seventh day the infant developed a pneumococcal pharyngitis with fever, which readily responded to chloramphenical p.o. During this period of stress she was lethargic and occasionally comatose. On one occasion the respiration became very shallow and finally ceased, necessitating artificial respiration for a short period. On the ninth hospital day re-investigation showed: serum Ca 4.89 mEq./1.; serum P 2.8 mEq /1.; blood serum Ca 4.89 mEq./1.; serum P 2.8 mEq./1.; **Blood Sugar** 15.9 mgm.%. Immediately 7 cc. of 25% glucose was injected i.v. with a dramatic response. Within 5 minutes the child was recovered from her coma, cried loudly, and sucked vigorously on a bottle. Blood sugar 2 hours after the injection was 45 mgm.%.

Further investigations: (1) A normal blood sugar and eosinophil response to s.c. epinephrine. (2) The same response to ACTH. (3) Oral glucose tolerance test fasting 8 mgm.%, 1 hr. 70 mgm.%, 2 hr. 45 mgm.%. (4) serum K - 5.72 mEq./1.; chloride 105 mEq./1.; Na - 134 mEq./1. (5) Serum cholesterol and liver profile normal.

The infant was started on 4 units of ACTH/kilo. in divided doses OH6. Repeated fasting blood sugars on this medication were 38.8 and 46 mgm.%. However, as soon as the dose of ACTH was reduced to 1/4 the initial dose, convulsions recurred (blood sugar 40 mgm.% at the time). The ACTH dosage was raised to the original level, and there have been no further seizures since Mar. 1, 1956. Fasting blood sugars on this dosage (Mar. 2) 55.4 mgm.%, with corresponding C.S.F. sugar 28.6 mgm.%.

This infant's mental status is questionable, and she appears to be blind. It is felt that she has suffered brain damage, probably due to the prolonged period of convulsion associated with her hypoglycemic state. It is planned to continue ACTH therapy, with gradually tapering dosage, and perhaps to change to oral adrenal steroids, (e.g. "Meticorten").

Case 2: H. S. Age 11 months. (Case of Dr. M. McLandress).

This firstborn female child, weight 8 lb. 3 oz. at birth, normal pregnancy and delivery. No abnormalities were noted in development or behaviour in the first 6 months.

In August 1955 she had an attack of agitation with frightened crying and pallor, followed shortly by blank staring, losing contact with her environment, and occasionally a few generalized twitching movements. During the next 5 months she had about 20 of these attacks, usually on awakening in the morning or after her afternoon sleep. On two occasions she became unconscious for a few minutes. Her mother claimed that the attacks were relieved by enemas (given for constipation).

On Jan. 26, 1956, the infant wakened early in the morning, became unconscious, and lapsed into a seizure with tonic and clonic movements, cyanosis, and involuntary defaecation, but no fever. On admission to Children's Hospital, she was lethargic (sedated at home), but examination was completely negative. As the sedation wore off, she became increasingly irritable when disturbed. Fluids were taken well, solids poorly. Large doses of sedatives were given because of the extreme irritability, but discontinued in 48 hours because of drowsiness. On the third hospital day the child was found comatose at 7.30 a.m. She had occasional twitching of the extremities, profuse perspiration, and could not be roused. After a few hours she roused from her comatose state, but

for the next 48 hours she fluctuated between extreme irritability and extreme lethargy. EEG was reported as showing a high voltage with widespread slow activity. A random blood sugar was 34.8 mgm.%, and a fasting blood sugar (3 hrs.p.c.) 41.5 mgm.%.

In further investigations, serial EEG's were done a.c. and p.c., with reports of slow wave activity before feeding and normal activity ½ hr after feeding. Concurrent micro blood sugars were 40.9 mgm.% and 77.6 mgm.%. Serum calcium and phosphorus levels were normal. The child was started on a high protein diet which seemed to relieve the irritability and lethargy to a considerable degree. At this time a blood sugar 1 hour p.c. was 81.5 mgm.%. The child was discharged on dietary management with no further study because of difficulties with the parents.

She was re-admitted on Feb. 13, 1956, after having become more irritable and more difficult to feed at home (although she had no convulsions). Her blood sugar and eosinophil response to epinephrine was normal, as was a liver profile. On Feb. 15, she was started on ACTH 10 units OH 6, with a good clinical response, less irritable, less perspiration, and taking feedings well. Fasting blood sugar was 83 mgm.%. On Feb. 20 she was changed to ACTH Gel 10 units OH 12, and discharged two days later. Subsequent fasting blood sugars have been 86.8 mgm.% (Feb. 27), and 84 mgm.% (Mar. 9), and clinical course uneventful. It is planned to slowly reduce the dose of ACTH, depending on her clinical response.

Case 3: R. P. Age 2¼ yrs. (Case of Dr. M. Marmar).

This firstborn male child weighed 4 lb. 8 oz. at birth, normally delivered and rated Apgar 10. He had considerable difficulty in the first ten days of life, with cerebral and pulmonary signs. Clinically, he was thought to have a virus encephalitis, and 80 lymphocytes/cu.mm. were found in his spinal fluid. The only spinal fluid sugar estimation during this time was reported as "below 40 mgm.%." No blood sugar measurements were made. The infant was discharged on the 15th day, clinically well and weighing 5 pounds.

He was perfectly well until the time of his second hospital admission with a normal developmental picture. On Nov. 24, 1955, he awoke normally, refused breakfast (which was not unusual for him), and suddenly screamed, lapsed into coma, and began to convulse. The convulsions failed to respond to sedation after admission to St. Boniface Hospital. Examination showed a comatose convulsing 2-year-old, with no other findings, including fundoscopic (rectal temp. 97°). A spinal tap showed clear fluid with no cells, and normal chemistry except for a sugar of 19 mgm.%. Blood sugar—20 mgm.%. WBC was 19,000 with 90% polymorphs; BUN—30 mgm.%. CO_2 combining power 14 mEq./l.; urine had 4+ acetone and 2+ diacetic acid; X-rays of skull and long bones were negative.

By 12 hours after the onset, an i.v. was established and 25 cc. of 50% glucose injected. After 5 cc. the comatose child was moving and talking, after 10 cc. he was moving quite vigorously, and before the 25 cc. had been injected he was sitting up and trying to pull out the i.v.! Thereafter his clinical course was uneventful, with continued i.v. therapy. Further investigations, including liver function tests, eosinophil depression test (epinephrine), oral and i.v. glucose tolerance tests were normal. An EEG was reported as showing "gross slow wave abnormality in all leads, with no fast potentials seen . . . compatible with hypoglycemia."

The child was discharged well on a diet with supplementary sugar 11 days after admission. He was readmitted 3 days later, having had mild diarrhoea for 2 days at home. An immediate "enhanced" blood sugar was 36 mgm.%, although the patient had no signs of hypoglycemia. The following day a non-fasting sugar was 62 mgm.%. On Dec. 16, 1955, he was started on ACTH 10 mgm. OH 6, and 4 days later changed to prednisone ("Meticorten") 2.5 mgm. p.o. twice daily. An EEG on Dec. 23 was reported as showing "improvement" over the previous record, with some fast spike potentials seen.

He was discharged well on Meticorten, and has been clinically well at home since. His Meticorten dosage has been reduced to 1.25 mgm. b.i.d. A fasting blood sugar on March 10, 1956 was 74.9 mgm.%, at which time both his mother's and father's fasting blood sugars were within the normal range.

The varied clinical picture is evident from the case histories presented. The mechanism of production of the syndrome is not understood—in fact, the factors governing maintenance of a normal blood sugar have not all been clarified.

The theory that overproduction of insulin causes this hypoglycemic syndrome has, of recent years, been discarded. The place of glucagon in carbohydrate metabolism has not been clarified as yet. In spite of the inability to explain the syndrome, proper treatment before irreversible brain damage has taken place will assure a good prognosis.

In the management of idiopathic spontaneous hypoglycemia the following methods of treatment have, at one time or another, been tried:

(1) Dietary: There are reports of the use of high protein, low carbohydrate diets in the management of hypoglycemia. These have not proved satisfactory.

(2) Alloxan: In 1948 Talbot et al reported the trial of this substance in an infant with spontaneous hypoglycemia with some success. They suggested cautious further trials, but there are no further reports of its use. The hazards of Alloxan

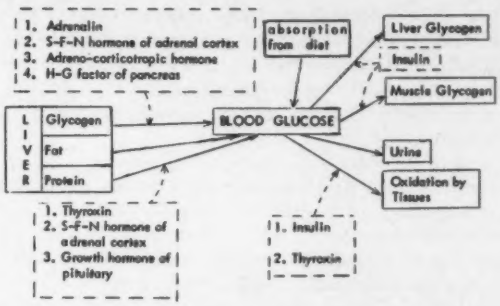
and the emergence of better methods of treatment make it advisable in therapy.

(3) Pancreatectomy: Pancreatectomy has been tried many times for treatment of the hypoglycemic symptoms, almost always with only temporary relief of a few weeks' to a few months' duration.

(4) Glucagon: This is a protein polypeptide presumably produced by the alpha cells of the pancreas, and closely allied chemically to insulin. It produces hyperglycemia by a direct glycogenolytic action on the liver. Straub et al purified and crystallized glucagon in 1953. Carson & Koch, and Schwartz et al report on its use in treating spontaneous hypoglycemia in children. Its action would seem to be of too short duration, and adrenal steroids seem to be more effective.

(5) ACTH and adrenal steroids: McQuarrie first discovered the value of these substances in the therapy or idiopathic spontaneous hypoglycemia of infancy. His suggested dosage is ACTH 4 mgm./kilo body wt./day, tapering the dosage while maintaining a symptom-free state and fasting blood-sugar levels of 40-100 mgm.%. Cortisone can be used equally effectively and more easily because of oral administration.

The prognosis for this syndrome is felt to be good, since the signs and symptoms disappear as the child gets older, and as the dosage is tapered,



but treatment for months or even years may be necessary.

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Psychiatry

The Importance of Social Incompetence in the Differential Diagnosis of Mental Defect

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Since certifiable mental defect is characterized by both intellectual inadequacy and social incompetence it requires to be distinguished from other conditions where either of these criteria might be present. This involves consideration of three groups of conditions[1]. The first group, marked by intellectual inadequacy alone, takes in intellectual retardation without social incompetence, intellectual dullness, and educational retardation or backwardness. The second group includes specific defects of a visual, auditory or speech nature which may be mistaken for intellectual inadequacy. The third group, distinguished by social incompetence alone, contains a number of disorders where aberrant conduct sometimes gives rise to the suspicion of mental defect. The extent to which this can occur may be illustrated from the first 250 cases referred to mental deficiency clinics in Scotland[2]. These consisted of 104 clearly certifiable mental defectives, 89 cases with intellectual inadequacy alone, 7 with specific defects, and 50 with social incompetence alone.

The group with social incompetence alone includes a considerable number of conditions ranging from plain maladjustment to juvenile general paralysis. Maladjustment not uncommonly leads to aggressive acts against the community, and occasionally it may result in referral for suspected mental deficiency. Referral, however, is more likely to occur in unstable adolescents. A certain amount of emotional upset is not uncommon in adolescence, and in some instances it may be of marked degree. In such cases the ardent desire for the limelight, the uncontrolled impulses, the recklessness and want of foresight may closely resemble the social inadequacy of the mental defective. This state is much commoner in females and its prevalence represents a serious social problem. Such girls are usually between 13 and 17 years of age, and not infrequently come before the courts charged with theft or sexual offences, or because they are beyond parental control. Although many have an intelligence quotient within the 70-85 range, the basic factor is not subnormal intelligence but loss of emotional con-

trol. Differentiation from mental defect may present difficulty, but in adolescent instability the condition is of recent onset with a relatively stable earlier phase of development, and a native level of endowment apparent on appropriate tests of intelligence.

A more extreme degree of abnormality is encountered in psychopathy, marked by persistent abnormality of character, episodic outbursts and antisocial conduct. Persistent abnormality of character is important in distinguishing this state from the preceding. The abnormality in psychopathy is habitual, present from childhood or early youth, and without response to training, punishment or any ordinary methods of treatment. Unlike the unstable adolescent the psychopath is seldom distinguished by redeeming features like generosity or thought for others. His outstanding traits are more likely to be selfishness, callousness and absence of shame. Often he appears incapable of observing or even understanding basic moral and legal codes. At the same time his transgressions reveal poor judgment where his own welfare is concerned. It is scarcely surprising that the question of mental defect should arise, and indeed some psychopaths have been certified under the Mental Deficiency Acts.

The possibility of mental defect may also require consideration in connection with epilepsy, brain-tumour and convulsive tic. In epilepsy the true level of intelligence may be masked as the result of fits or owing to dullness from the drug used. Failure to make allowance for these factors could result in a false diagnosis of mental defect. Apart from idiopathic epilepsy fits may occur in cerebral tumour, and, where this is associated with deterioration in conduct and intellect, mental defect might be suspected. Such a suspicion may also arise in convulsive tic or Gilles de la Tourette's disease[2]. Since tics are of very frequent occurrence in low-grade defectives, it is not surprising that a diagnosis of mental defect should be entertained in convulsive tic with its widespread involuntary movements, inarticulate cries and coprolalia. The disease usually begins at an early age with involuntary movements of an arm, shoulder or face, and these increase in extent and intensity until the entire body is involved. Such movements include twisting of the head and neck, kicking and jumping, and grinding the teeth. Involuntary cries come later, and as the disease advances there appear echolalia, echokinesia and finally the pathognomonic coprolalia.

Difficulty in diagnosis may arise in the schizophrenias, Heller's syndrome and juvenile general paralysis. Amongst illnesses of a schizophrenic type the degree of intelligence is liable to be suspect in early infantile autism, where from the beginning the child fails to make the usual response to people. He may or may not learn to speak, and even should he acquire this ability, he still does not speak spontaneously. When he responds to questions, his meaning con only be elucidated with difficulty. Nevertheless from the irrelevant and metaphoric answers the impression may be conveyed that the child understands the nature of the question. Thus, to the question "What is an apple?" one autistic child replied with the word "orange." Such a child pays no attention to the people around him, but will carry on his activities in an apparently purposeful fashion with the things in his environment. Children with this condition are not uncommonly regarded as deaf or defective, and an accurate estimate of intelligence is difficult or impossible owing to inaccessibility. The true state of affairs, however, is suggested by the contrast between the limitation of speech, stereotypy of behaviour and indifference to people on the one hand, and the alert, intelligent expression and quick, well coordinated movements.

Whereas the preceding condition is present from the first year, illness of a schizophrenic nature may appear later in childhood with the onset of excessive day-dreaming, diminution of interests and reduction in physical activity. At the same time there is likely to be a growing seclusiveness associated with irritability when interfered with, sensitivity to criticism and bizarre behaviour. The older the child the more the pattern approximates to the adult type. Confusion with adult schizophrenia is most likely to occur during the development of the simple-hebephrenic variety. The mental inertness, poor work record and impulsiveness may be mistaken for mental defect. As a rule there is a falling off from previous standards, but, when schizophrenia develops in dull individuals with an inconclusive earlier history, doubt is apt to be encountered.

In advocating the diagnostic value of the Arithmetic, Similarities and Block Design tests of his adult intelligence scale, Wechsler[4] points out that schizophrenics may do well on any or all of these, in contrast to the performance of mental defectives.

In Heller's syndrome or dementia infantilis, a condition originally thought to be schizophrenic, but now regarded as an organic degeneration[5], speech is rapidly lost and replaced by inarticulate grunting. Associated with this is a state of overactivity characterized by gesturing and grimacing, tic-like movements and bizarre posturing. In spite of these changes affected children are said to retain an intelligent facial expression. Finally, consideration should be given to juvenile general paralysis, which, when of simple dementing type may simulate mental defect.

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Bacteriology

The Clinical Application of Bacteriology

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At first glance this would appear to be a very simple procedure. All you do is collect a specimen from the patient and ship it off to the Bacteriological Laboratory, then treat the patient symptomatically. A mere trifle, and one whose details are not worth concerning one's self over. The only nuisance about the procedure is filling out the necessary form. For some obscure reason, bacteriologists want a lot of information about the specimen sent them, and they also seem to be inordinately fussy about the way their specimens are taken and handled. Why can't they be like urologists and haematologists who never complain about the specimens they get? But the bacteriologist practically wants a running commentary on the case in hand. Since it is, probably, just idle curiosity upon his part, the best way to handle the situation is to mystify him. Fill out the form with a leaky pen, or better still a scratchy ball-point, in a professional scrawl, especially designed for impressing patients with the obscurity of their ailments and keeping pharmacists on their toes.

Perhaps, what I have to say in the following will explain in some measure just why bacteriologists are so insistent about mere details, and require so many facts.

Now, to express it in the simplest terms possible, there are just three good reasons why the clinician bothers to send specimens to a bacteriologist for examination at all: to find out whether there is any unusual change in the type or distribution of the usual bacterial flora, if some abnormal flora could be responsible for the patient's symptoms, and, if so, its sensitivity to antibiotic agents. Usually, it is a fairly simple matter to answer the first and last of the three questions raised, but it should be understood by the clinician that the second is difficult, and sometimes practically impossible, to give an adequate reply to.

Suppose, for example, that the bacteriologist finds a certain organism, say a Staphylococcus, which in his perverse way he insists upon calling Micrococcus, probably just to confound the medical profession. Now, is it the reason for the patient's illness? Arguing from statistics the bacteriologist may say that since the patient's disease is usually caused by a Staphylococcus, then this is most likely to be the case here too. Sad to say, this is an argument which does not always stand up. Every physician knows that two patients can never be counted upon to react exactly in the same manner to the same infection, and, what is still more important, virulence may vary considerably between strains of bacteria even though they may be of identical species. There is really very little difference between Micrococcus pyogenes var. aureus and var. albus besides pigment production, but the former is frequently more pathogenic. Using the argument from statistics, since it has long been taken for granted that Streptococcus pyogenes is a cause of severe sore throat, then we assume that in all cases of sore throat where Streptococcus pyogenes is found present it must be the causative organism. But this could be, pardon the expression, dead wrong. Bacteriologists know that Streptococcus pyogenes is frequently found in the throats of quite normal, healthy persons. It is within the realm of possibility that such an individual could develop diphtheria, so, then, if we blamed the sore throat upon Streptococcus pyogenes we would be sadly mistaken, and the pathology department might well have another customer.

It should be recognized by the clinician that micro-organisms frequently have Jekyll and Hyde personalities. The same organisms may be, at various times, either a vicious pathogen or a harmless saprophyte. There is no doubt in any one's mind that when a Micrococcus produces furunculosis it is a pathogen, but when it is behaving itself, as it usually does, it is just another free-loading boarder on the skin. The presence of a pathogenic micro-organism, on or in human tissue, may result in any one of a number of conditions, ranging from the carrier state to candidacy for the autopsy table. It is unfortunate, but we have not to date learned just what the factors are that will determine which of its two personalities it will manifest, and, if it should be the pathogenic one and infection arises, at just what stage of the illness an equilibrium will be reached in the host-parasite relationship. It has been known from of old that certain infections confer a lasting immunity against their recurrence, and, in the light of modern medical knowledge, it is readily explicable why, although the patient is carrying the organism or has come in contact with it, no symptoms of infection appear. We know that with some diseases certain conditions must be fulfilled before the infectious agent can gain a hold. This explains why the finding of Clostridium perfringens (we used to call it Welchii) in a wound does not necessarily predicate gas gangrene. However, specific immunity and special growth factors and conditions are not always involved when there is other than the usual response to the presence of some organism in the tissues. In these cases we have to

suppose that either the organism is unusually virulent or the patient's resistance is very low.

Just as a bacteriologist cannot summarily dismiss an organism as a mere contaminant because it is not usually considered to be pathogenic, he cannot accept a micro-organism as the essential cause of a disease simply because it usually is.

If we return for a moment to our original argument about the Micrococcus and the supposed micrococcal infection, it would seem that the simplest solution to the difficulty, assuming that we had the necessary time for experimenting, would be to apply the test of Koch's postulates. Although the good doctor did not actually formulate them as such, the postulates are in brief, that if you can always, and in every case, isolate a certain micro-organism, and that if you can grow it in pure culture for several successive transfers from medium to medium, and then reproduce the same disease in some susceptible host with it, then, it is the cause of the disease. Unfortunately, Koch's postulates cannot always be fulfilled, either because there are no susceptible hosts that can be used or because the organism cannot be cultivated upon laboratory media. An alternative method of demonstrating the organisms culpability must be sought.

As the casual role of a bacterium in a disease may be more logically assumed if there is a rise in antibody titre to it in the patient's serum during the course of the disease, followed by a fall in this titre after recovery, bacteriologists sometimes request several blood specimens for serological examination during the course of a patient's illness.

Doubtless, the clinician sometimes wonders just what the bacteriologist does with the specimen he sends him. In many cases a few rapid and simple tests are performed upon it which, when considered together with the case history and physical findings, will help the clinician establish his diagnosis. A common illustration of this sort of presumptive test is that if sputum be taken from a patient with symptoms of pulmonary tuberculosis, and if it be found upon examination by acid fast staining technique to contain acid-fast bacilli, then it is quite likely that the patient has pulmonary tuberculosis. Another example might be that, if Gram negative cocci are found in cervical smears taken from a woman suffering from an acute infection, gonorrhea would be suspected. However, it cannot be too deeply impressed upon the clinician that this is purely presumptive evidence, since organisms such as these may be found in normal, healthy individuals and that they are not necessarily the pathogens which immediately spring to mind. There are many species of quite harmless, saprophytic micro-organisms which fit the above descriptions. Therefore the diagnosis must be an essentially clinical one unless further, more determinative, tests are carried out upon the specimens.

When, as is usually the case, the tests are carried further, the clinician has the evidence of the very highly probable pathogenicity of the organisms based upon their isolation, cultivation and identification. For example, in the cases cited in the foregoing paragraph above, on the basis of simple examination they could only correctly be reported as "acid-fast organisms found present" and "Neisseria seen." Remember, please, that this means just what it says, no more. Acid fast organisms can be isolated from tap water or from off leaves of grass, and Neisseria from just about anybody's nasopharynx.

A type of problem that often arises, when only simple evidence is presented, is, that the clinician will think that the patient has tuberculosis, which he knows to be caused by an acid-fast organism, and consequently will expect the bacteriologist to find Mycobacterium tuberculosis present. The bacteriologist will find acid fast organisms present in the suspect's sputum and conclude that, since the clinician has indicated he thinks the patient has tuberculosis, then these acid fast organisms must be Mycobacterium tuberculosis, and is sorely tempted to report them as such. This, of course, is circular reasoning and the conclusion consequently quite invalid. In a similar manner, a small or insignificant rise in antibody titre to some bacterial or viral antigen may occur in the serum of a patient about the origin of whose illness the clinician is none too sure. If the clinician is unduly pressing for the bacteriologist's opinion, wishing to please, he may agree with the clinician that the rise in antibody titre might be considered significant, and so the patient may have such and such a disease. This may satisfy the clinician, since it agrees with his preconceived idea, but it is really worthless evidence and what is worse the bacteriologist probably knows it. In this connection, I should like to point out that, although laboratory tests are frequently repeated when their results fail to fit the clinician's expectations, it is not often suggested that they be repeated when they do fit. It's definitely not cricket to use a test for the diagnosis of a particular infection, and at the same time use the clinical findings to judge its reliability.

Now, of course, you may jump to the conclusion that it is best, then, to keep the bacteriologist completely in the dark concerning the clinical findings, so that he can give an unbiased opinion. That would be alright, but when used with discretion, knowledge of the patient's condition is very valuable to the bacteriologist. Although in all good laboratories the routine treatment of specimens is so devised, that no usual pathogen is likely to slip by unnoticed, still much

time can be saved in isolating the causative organism if the patient's clinical condition is known to the bacteriologist. If the patient's illness suggests that a specific organism may be involved, then the bacteriologist will use special media from the beginning of his investigation designed for the isolation of that micro-organism. As an example of this, if a swab taken from the wound of a patient suffering from apparent gas gangrene were sent to the laboratory together with a notation of the fact, then, although it would be found by the routine method, in time, special methods of cutivation and special media would be applied from the beginning of the investigation, so that the identification of a Clostridium could be made in less than half the time required by the routine method. Obviously this could be considerably advantageous to the patient.

The clinical bacteriologist normally has two courses open to him as regards the way in which he may conduct his investigation of specimens sent in for bacteriological examination. He may either identify in full all the organisms found present in the specimen, both as to genus and species, or he may indicate the genus only, when the specimen comes from a site normally sterile and this on the basis of rapid testing methods. Of these two courses, the first is rarely followed. It involves altogether too much time consuming work, and for practical purposes is rarely necessary. The latter course is most likely to be the one of choice, and, since specimens from normally sterile sites do not usually contain more than one type of micro-organism at a time, this does not involve too much lost time. The species is not necessarily given because it would require much time to ascertain, merely delay the report to the clinician, and in any case serve no really useful purpose. It should be obvious that specimens coming from sites which have a normal flora require only the identification of pathogens, or at least of potential pathogens, and it is customary to report only these to the clinician. Identification of the other organisms present is normally of purely academic interest.

It is, of course, understood that there is great urgency for speed in clinical bacteriology and, consequently, it is perfectly legitimate for a bacteriologist to use his p a s t experience in identifying micro-organisms by their morphologic appearance alone when giving the clinician his preliminary report. However, the work must not be permitted to end with such cursory examination, because the bacteriologist knows that there is a great tendency to morphologic variation amongst micro-organisms, even amongst strains of the same species, and identification on the basis of superficial appearance alone is neither adequate nor in keeping with his scientific integrity.

Since the discovery of antibiotics, one of the most important functions of the bacteriological laboratory has become the testing of micro-organisms for their antibiotic sensitivity. Indeed, this has become so much the case, that the clinician's chief interest has now become not so much to know what the infecting pathogen is, but rather what antibiotics it is sensitive to. It is well known to bacteriologists that in the presence of biologicals, either in vivo or in vitro, many micro-organisms undergo surprising morphological changes which render their species, and sometimes even their genus identification, very difficult by simple inspection alone. This is an excellent reason why routine methods should be based upon sound scientific principles, together with a knowledge of the various factors which affect bacterial growth. It is sometimes foolishly thought that academic and clinical bacteriology are quite far removed from each other, and, consequently, a clinical bacteriologist need merely be a technician knowing a few tricks for the rapid identification of pathogens who should leave the more fundamental knowledge to the academician. This is definitely a false premise for the clinical bacteriologist who is not sufficiently trained or fails, from lack of understanding of the problem he is up against, to apply the scientific method will fail to recognize departures from the accepted host parasite relationship with possibly fatal results. Of course, it may be argued that such observations as he might make in the course of his routine investigations are unimportant in the treatment of the individual patient—which is true—but it is, nevertheless, upon him that the growth of our knowledge of infectious diseases depends.

A point which should be impressed upon all clinicians, is that the results of the investigation of clinical material, and the speed with which such results are obtained, do not depend solely upon laboratory routines but very much upon the manner in which the specimens submitted for examination to the bacteriologist have been taken and upon the promptness with which they are transmitted to the laboratory.

It cannot be too greatly emphasized that the sampling of fluids, such as urine and cerebrospinal fluid, which are normally sterile, demands extreme care upon the part of the sampler to avoid contamination. Even greater precautions are necessary than are taken in the operating theatre. This may seem like a rather exaggerated statement, but it should be recalled, that, although aseptic surgical technique aims at the prevention of contamination of open wounds by pathogenic micro-organisms, it takes very little account of contamination by air-borne saprophytes. An entire investigation can be ruined for the bacteriologist by the chance entry into a culture of even a single organisms not originally present in the clinical specimen. Of course, specimens from sites such

as the upper respiratory tract, alimentary canal or vagina, which have a normal flora, may be taken without too great precautions, but even here, the introduction of extraneous organisms should be avoided as much as possible. It should be remembered that what the clinician wants to know is not what organisms he has, but what the patient has.

It does not seem to be understood by clinicians in general that delay in sending specimens, he has taken, to the laboratory can make it quite impossible for the bacteriologist to cultivate pathogens which may have been present originally in the material. Many pathogenic micro-organisms are extremely delicate, strange as it may seem, and they may die not only from lack of their proper requirements such as essential nutritives, temperature and pH, but also from the effects of such factors as desiccation, antiseptics, bactericidal body secretions and enzymatic activity. When saprophytic micro-organisms are present, either as contaminants or originally introduced together with the pathogen, being made of sterner stuff they not only survive, but also multiply at room temperature often to such an extent as to completely obliterate the pathogen before its culture can be undertaken.

It is suggested that the following general precautions should be observed when taking specimens for examination in the bacteriological laboratory:

Attached to all specimens should be a clearly printed record of the patient's name, number and ward, the nature of the specimen, the site from which it has been taken, the clinical diagnosis and duration of the patient's illness, antibiotics administered previous to taking of the specimen, and, finally, but most important, what examination is required of the specimen, such as identification and sensitivity to biologicals. If specimens are to be examined serologically for the presence of antibodies to viruses, the specific variety of virus for which the clinician desires the examination should be clearly stated. "For viral studies" is not sufficient, because there are so many possible viruses the specimen could be titrated for, that a complete examination, on that basis, could involve so much work for the serologist that the patient would be either dead or recovered by the time the examination was completed.

Specimens which have been taken for culture should never be permitted to come in contact with antiseptics or disinfectants. Skin scrapings taken from an area which has already been treated with anti-fungal or other ointments are usually worthless for culture. If a site must be cleansed before the specimen is taken, this operation should be performed only with a sterile cotton swab moistened, if necessary, only with sterile physiological saline. The swab would be best held with sterile forceps rather than the fingers.

All specimens taken for culture should be sent to the laboratory without any delay. Remember that it is frequently impossible to cultivate micro-organisms from desiccated swabs. Speed is of the essence in the following investigations:

Cerebro-spinal fluid: since N. meningitidis and N. gonorrhoeae are both extremely delicate organisms and sensitive both to oxygen and cold, fluids suspected of containing them should be kept at body temperature from the moment they are taken and sent to the laboratory immediately for examination.

Eye-swab cultures: since the lachrymal secretions contain lysozyme, which is rapidly fatal to bacteria, such swabs should be plated out immediately upon taking.

As regards the precautions which should be taken concerning other specimens:

Urine: should be taken by catheter, and with strict aseptic precautions, directly into a sterile flask, however, clean, mid-stream urine is suitable for culture purposes if taken with care from male patients.

Faeces: should always be collected in sterile bedpans. There are enough things in faeces as it is without compounding the bacteriologist's difficulties. The whole stool specimen need not be sent but a part of it containing any abnormal matter, such as pus or mucous should be placed in a sterile container for shipment to the laboratory.

Sputum: Should always be collected in a sterile container when the patient first awakes in the morning. If the patient cannot produce much sputum readily he should be posturized, with head and shoulders lower than the chest. After some ten minutes in this position usually sufficient sputum drains into the trachea for the patient to cough it up into the sterile container.

Serous fluids: pleural, pericardial, synovial and ascitic fluids should be taken with strict aseptic precautions into sterile containers and sent at once to the laboratory.

It is usually more convenient for all concerned if blood samples are taken by a laboratory technician. If the patient's condition is one in which there is a regular rise in high temperature then the specimen should be obtained just as the temperature begins to rise. Blood specimens should be taken with a sterile syringe and needle after careful cleansing of the site of venipuncture with alcohol and iodine. The contents of the syringe, usually 10 cc, is immediately placed in a sterile tube containing an anti-coagulant. This is essential because if the blood is permitted to clot

many organisms may be trapped in the fibrin mesh and become unavailable for culture. An alternative method is the use of a disposable vacuum ampoule and needle, such as the Keidel tube, which contains the anti-clotting agent. The specimen should be expressed to the laboratory at once.

On the whole, medical men generally do not seem to realize that, when blood is taken for culture, there may be very few viable organisms present in the specimen. Even in severe bacteremia there may be only one or two organisms per cc. in the circulating blood, and sometimes even fewer. Just to make things more difficult, among the bacteria species which may be found in the circulating blood there are several which can be grown upon laboratory media only with considerable trouble. Commonest amongst these are members of the Parvobacteriaceae, such as Brucella and Haemophilus. This does not mean to say, of course, that only these organisms may be found in the blood because numerous species and organisms, both pathogenic and normally saprophytic, have been reported found in the blood at one time or another.

If any bacterium at all, no matter how harmless, is recovered from a body fluid known to be normally sterile, such as the blood, cerebro-spinal fluid, or urine, the bacteriologist must regard it as a potential pathogen. He has an obligation to both the clinician and the patient to report its presence and cannot merely dismiss it as a probable contaminant, even though he may be morally certain that it is.

It should not surprise the clinician if the laboratory reports a number of different kinds of organisms found together in pleural and peritoneal fluids; these may be even mixtures of both aerobes and anaerobes. This is quite the usual state of affairs. However, with the other body fluids it is usual to find only one species of micro-organisms present at a time, yet it must be borne in mind that mixed infections do occur even in them, although rarely.

Now, having rapped the clinician's knuckles on matters bacteriologic, what sort of co-operation can he expect from his laboratory confreres? The answer to this depends not only upon the suitability of the specimen, which, to some extent, at least, is in the hands of the clinician, but upon the time and facilities available for the research required. However, he may expect this: first, the isolation and identification of known pathogens; second, information as regards the sensitivity, if any, of the pathogen in question to antibiotics. With the exception of extremely slow growing micro-organisms, such as Mycobacterium tuberculosis, this information should normally be available within 48 to 72 hours after the specimen arrives in the laboratory, and often may be had in less time than that. In addition he may expect co-operation and advice when requested, the value of which depends upon the bacteriologist's training and integrity.

St. Boniface Hospital

Consultation

Special Report from Panel Discussion by Members of Courtesy Staff of the St. Boniface Hospital

A very entertaining panel discussion on "Consultations" was held by the Courtesy Staff of St. Boniface Hospital on April 10, 1956. The members of the panel were: Doctors W. Albi, P. Doyle, S. Guslits, S. Golfman. The topic was discussed under the following heads:

1. The proper way to ask for a Consultation.
2. The proper conduct of a Consultation.
3. What about compulsory Consultation?
4. Are Consultations always beneficial to the patient?

The subject was studied in a very lively manner. Several questions were collected from the audience and answered by the panelists.

The main points brought out are hereby summarized:

1. The following terms were defined: Consultation, Consultant, Attending Doctor.

By a Consultation is meant the co-operation between practitioners in the formulation of diagnosis, prognosis and treatment of a given case. The practitioner consulted is the Consultant. The practitioner seeking consultation is the Attending Doctor.

The practitioner consulted is usually a specialist, if the case in question is an obscure one. If the consultation is obtained for the purpose of confirmation, the consultant may be of less experience than the attending doctor.

2. If the patient is to be sent to the consultant's office, the attending doctor shall send along with him a letter, containing the significant details of the case. After examining the patient, the consultant shall forward his opinion in a sealed envelope to the attending doctor. He may give the patient such information as he judges appropriate to his position.

3. If the patient is to be seen by the consultant and attending doctor together, the following procedure is generally adopted:

(a) the parties should be punctual. If the consultant is late, the consultation may be cancelled after a reasonable wait. If the attending doctor is late, after a reasonable time, the consultant may see the patient, and communicate his findings in writing to the attending doctor.

(b) the attending doctor should enter the patient's room first to introduce the consultant and leave the room last to assure that someone is left with the patient.

(c) the diagnosis, prognosis and treatment should be discussed in private. The opinion on the case and the treatment as agreed shall be communicated to the patient, when practicable, by the consultant in the presence of the attending doctor.

(d) it is the duty of the attending doctor to carry out the measures agreed upon. He may alter these measures only in an emergency or after adequate trial.

4. The attending doctor is responsible for the conduct of a consultation. If he conducts the consultation well, he will improve his position with his patient and with the consultant. If he conducts a consultation poorly, he runs the risk of losing his patient.

5. If the consultant and attending doctor hold divergent views, the patient, or his representatives, should be acquainted with the difference of opinion. The patient, or his representative, then shall decide on one or other of the suggested alternatives.

6. The nursing staff at the hospitals should be reminded that the recommendations left by a consultant are not orders and that the recommendations shall be carried out only after the attending doctor has given his consent. The nursing staff should also be reminded that when further orders are needed, they shall call the attending doctor and not the consultant. If the attending doctor wishes the consultant to leave orders, he should indicate it clearly on the chart.

7. One member of the panel brought out the inadequacy of the consultation forms available at St. Boniface Hospital. For complicated cases a separate form should be available with sufficient space to permit the attending doctor to write in all the significant details.

8. In certain situations, a consultation is compulsory. It was brought out by the panel that compulsory consultations at St. Boniface Hospital were not overdone. These compulsory measures serve to protect the patient, the attending doctor and the hospital.

9. Certain cases were mentioned where the patient did not benefit from the consultation. It was felt that the fault lay with the attending doctor in his failure to choose the proper time for consultation, or the proper consultant, or in his improper conduct of the consultation.

10. The panelists agreed that the majority of consultations were well conducted. However, there was room for improvement:

(a) The need for a consultation should be anticipated by the attending doctor. The attending doctor should accept the opportunity of a consultation in obscure cases. He should acquiesce in any reasonable request for a consultation by the patient.

(b) The attending doctor should write out the consultation sheet (or the letter) setting out in detail (and in order) the essential information.

(c) The attending doctor should indicate clearly what he wants the consultant to do. Report as to diagnosis, report as to treatment, take the case over.

(d) The consultant should avoid trying to "steal the show"; by words that would undermine the patient's confidence in the attending physician; by visiting the patient after the consultation is over and without the attending doctor's knowledge; the attending doctor should avoid words that would make the patient doubt the consultant's advice.

11. (a) The modern scene shows the general practitioner and the specialists in sharp competition for the patient's attention. This situation tends to make the general practitioner reluctant to call a specialist in consultation. This is unfortunate. We are all agreed that both general practitioners and specialists are essential to a sound medical service. We all need help at times. People today are aware that there is a natural limit to a physician's ability.

(b) We should strive to maintain a sound approach to consultation. We should conduct a consultation well and adhere to the rules of medical etiquette. By so doing, we will improve our positions with the patient and with the consultant.

Obituary

Dr. Livingstone Gilbert Gunne

Dr. Livingstone Gilbert Gunne, 66, V.D., died on August 7 at his home in Lac du Bonnet. Born in Glenboro, Manitoba, he graduated from the University of Manitoba Medical School in 1913. He served with the British army in France in the First World War, winning the Meritorious Medal and life membership in the Canadian Legion. For thirty years he practiced with his father, Dr. W. J. Gunne in Kenora, Ontario. There he was head of the Medical Board and an officer of the 16th Medium Battery. He held the rank of Lieutenant-Colonel. After his service in France he was in China for a year, examining coolies for a labor corps; then was acting commanding officer of Fort Osborne Barracks, Winnipeg, during the influenza epidemic. The last seven years of his life were spent at Lac du Bonnet.

Abstracts from the Literature

The Hazards After Cholecystectomy, with Review of 500 Case Records. Yeats, J. M. M. J. Australia, 1: 646 (Apr.) 1956.

500 consecutive cholecystectomies were reviewed, covering 1949 to 1954. The common duct was explored in 51 cases, stones being found in 23. Post-operative complications included eight patients with serious pulmonary disease (mostly collapse); 5 with diagnosed venous thrombosis; 10 with hemorrhage in the wound; 3 with prolonged bile drainage; and 21 with wound infections sufficient to keep the patient in hospital over 14 days; 4 cases with rupture of the wound. There were no pulmonary emboli. The mortality rate was 1.1% after cholecystectomy alone. All deaths were due to a lethal pool of blood, bile or pus. After choledocholithotomy (22 cases) the mortality rate was 18.2%. Three of the 5 deaths were due to leakage of blood, bile or both. Serious complications, including deaths, rather than to the heart, and other areas remote from the operation site. It is important to treat the cause of the collapse, and not the effects. It is mandatory to remove the gallstones soon after their discovery, as the serious complications tend to occur in older patients who have had the stones for a long time.

A. G. Rogers.

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A Comparative Study of Three Anticholinergic Drugs - Monodral, Pamine and Pro - banthine. McKenna, R. D., Bourne, R. H. and Arendt, E. Canad. M.A.J., 74: 685 (May) 1956.

The effects of single doses of Monodral, Pamine, and Pro-banthine on basal gastric secretion in patients with radiologically proven peptic ulcer was studied. The effects on the volume, free acid, and pH were studied in patients with fasting free acid of over 20 clinical units. 177 studies were performed on 116 patients. 5 or 10 mg. of Monodral is an excellent suppressor of volume and acidity of the basal gastric secretion. It is as effective in hypersecretors, as in those with less active secretory reponse. Pamine and Probanthine, in the doses studied, were not as effective as Monodral. Probanthine can be absorbed from rectal suppositories. Side effects were less frequent and less severe with Monodral and Probanthine.

A. G. Rogers.

Editorial

S. Vaisrub, M.D., M.R.C.P. (Lond.), F.R.C.P. (C.), F.A.C.P., Editor

The Soft Underbelly

Medicine is a beleaguered fortress. It is a citadel besieged by armies of public spirited citizens waging a holy war on "professional privilege" in the name of the "people" clamoring for the attainment of the utopia of "State Medicine."

In its defence against the threatening danger, the medical profession depends largely on the persuasive powers of its Public Relations program. The latter aims at creating a better understanding between patient and doctor, as well as convincing the public that they have a great deal to lose and very little to gain from handing over their health to the Government. It emphasizes, as it should, the concern of the doctors with the welfare of the people, as well as their health.

It may well be, however, that in presenting his case the doctor has been overplaying, somewhat, the "unselfishness" of his motives. Unselfishness is always suspect. Fed on exaggerated, misquoted and misinterpreted reports of the doctors' "average" income, the public is apt to ascribe the doctor's attitude, not to unselfishness, but to greed and are apt to question the sincerity of the doctor's professions to the contrary. Perhaps, the injection of a mere "selfish" note into the dispute may be advisable. Let the doctor, then, submit that, by and large, as an individual he dislikes regimentation and loathes bureaucratic control, and that he has not anticipated, when he undertook the arduous journey into Medicine that his will be the profession singled out for socialization—an island in the sea of free enterprise. Let the doctor state that, if he were to labor under conditions distasteful to him, he would not be able to do so as efficiently and enthusiastically as he has hitherto. Nor would he be likely to inspire his sons and ambitious youngsters of his acquaintance to follow in his footsteps. Acquainted with this aspect of the problem, the public may well hesitate before proposing anything which would radically upset the delicate patient - doctor ralationship by confronting them with disgruntled physicians with "chips on their shoulders."

An attempt is being made currently by the Dominion Government to begin the assault on free Medicine by employing the Churchillian strategy of attacking its "soft underbelly." The socialization of diagnostic services, as proposed in the National Health Bill is the first tactical move in this strategy, The Act provides for the nationalization of the seemingly marginal laboratory services not directly related to medical skill and knowledge, performed by non medical staffs of technicians. At first glance this would not appear to be an issue on which the doctors could take a stand, but on closer examination it is easy to see that the act represents an encroachment on medical premises. Laboratories may be staffed by lay personnel, but they are supervised by physicians. Moreover, a number of laboratory services are often performed by the doctors themselves, and, what is even more important, many tests such as electrocardiography, electroencephalography, radiography and others require interpretation by physicians who have devoted much time and effort to acquire the necessary knowledge. Apart from finding themselves shorn of a sizeable part of their income, many doctors will find themselves deprived of the satisfaction of being able to exercise their skill, unless, of course, they become full time government employees.

It would be a pity indeed if this bill and others of similar ilk gained publicity and passage, unresisted by the medical profession, thus marking a landmark in the retreat and ultimate defeat of free medicine.

Let us be on guard. Ed.

Letters to The Editor

The Doctor at the Wheel

Dear Editor:

The doctor of today lives with his fellows in a world of expanding horizons in almost all fields of human activity. His own field in particular is challenging him continually and sharply. The output of the scientific press defies his coverage; and the growth of the specialties is a natural sequel to the rushing tide of new knowledge.

Time, the fourth dimension in a curved space is perhaps the most inexorable factor in the existence of the doctor of today. It can no longer be familiarly taken by the forelock. In this age of tabloid reading . . . of P.A.S., B.U.N., P.S.P., ECG, M.L.D., N.A.D., V.D.H., and G.O.K., the doctor is making the unpleasant discovery that he can scarcely tolerate the dignified leisurely Victorian approach to character portrayal, nor the stately beauty of a Mozart minuet, nor the contemplative landscapes of Claude Lorrain. Such is the nature of the doctor's sharpened and newly conditioned reflexes.

His 225 H.P. coral and maroon juggernaut proclaims his success. Power steering, push-button driving. automatic windows, power brakes, tubeless tires—all these things have reduced his driving to a state very close to complete automation, except for one thing—the judgment of the driver. Some four people drive his car, each with a different reaction time, each with a different degree of attention: each with a different set of preoccupations: each with a different approach to the terrible varied traffic problems: each with a differently adjusted emotional thermostat.

It was my privilege to serve on a committee of such doctors last June. They came from St. John, Newfoundland to Victoria, B.C.; and the purpose of the assembly in the luxurious board room of the Montreal General Hospital was the commencement of a study of ways and means to minimize the toll of human lives taken by traffic accidents in Canada.

The chairman polled the meeting; and each member of the committee presented first blush ideas which almost ran the gamut of traffic legislation, police reform, safety gadgets, punishments, rewards, traffic engineering potentials, driver testings, driver elimination, casualty training for internes, hospital cooperation, research projects—medical, surgical, and psychiatric.

These men had been thinking good sound thoughts. Of course there were a few of whom Julius Caesar might have said: "He is a dreamer. Let us leave him. Pass ."; but there has been too much good fruit born of dreams for such to be ignored.

The Canadian provincial autonomy has developed individual thinking, and a good thing too, for in the matter of traffic problems each province has its climatic, geographical, geological, geophysical, and constructional factors, some of which are peculiar to each, though many are common to several or all.

It soon became apparent to us all that an approach to uniformity in traffic legislation was long overdue; but it was at once recognized that no collection of information was as yet available for practical study to be made.

It was equally recognized that any haphazard approach to the automotive executives in the matter of safety gadgets would not, nor indeed should not meet with enthusiastic acceptance. Although the trade at large accepts the principle of safety, from the salesman's point of view an accent on safety carries a sinister connotation of potential danger in this 225 H.P. lethal vehicle placed in the hands of the buyer.

Out of all this and much more came the realization that a long term statistical study of the many facets of this protean problem was an actual prerequisite to any practical advance. It was therefore decided to set up a foundation for the study of traffic accident problems: the participants to be the Canadian Bar Association, the Canadian Medical Association, and the Association Medical Francais.

The delegates, having launched the project, returned each to his province, local association, and committee with the request from the general committee to embark on the local problem study, so that when the statistical study is commenced, the local ground work having been begun will, perhaps, facilitate the progress toward a rather distant but most desirable goal—the saving of Canada's most precious commodity which is neither wheat nor minerals, nor fisheries nor forests, but Human Life.

On behalf of your local Committee On Traffic Accidents I would ask you, dear reader, (Passing by the editor is strictly against the rules of epistolary traffic! Ed.) to give us your assistance by:

(a) putting your ideas for the minimizing of traffic accidents into writing and sending them to Dr. M. T. MacFarland, the secretary of the local committee.

(b) setting yourself the task of personally becoming a courteous, careful driver.

(c) preaching traffic behaviour to your patients.

(d) observing the city and country as a self appointed inspector of traffic (without portfolio) in order to collect ideas.

(e) listening to your teenagers and your wife as they discuss traffic with you.

(f) being a friendly driver, not a quarrelsome, pontifical superior, in spite of that last traffic ticket and your smarting sense of cruel injustice or terrible bad luck ("of course it would happen to me") remembering that as Gilbert and Sullivan said: "The policeman's lot is not an 'appy one." He may have dyspepsia too. His "patients" are often most unreasonable and trying. He is not perfect, and his language may not reflect that sense of civil service which is the badge of the perfect gentleman. BUT he will, and does help in a hundred ways . . . and this is a true thing. I have seen his face as he picks up the dead child from the middle of the road. He is your fellow man, and you can make him your friend. Please do so.

As a member of the medical profession you are now a part of a fine movement facing a grim challenge.

Drive on, Doctor!

Athol R. Gordon, M.D.
Chairman,
Committee On Traffic Accidents,
(Man.)

Postgraduate Education for the General Practitioner

Dear Editor:

I would like to express my sincere appreciation to the Manitoba Institute for the Advancement of Medical Education and Research for making available a grant which stimulates postgraduate education among the general practitioners of Manitoba, and my thanks to the Committee appointed by the College of General Practice for naming me the first recipient of this excellent award. I elected to attend the Cook County Postgraduate School of Medicine in Chicago. This school was founded in 1932 by members of the attending staff of Cook County Hospital. It operates under the supervision of the attending staff who are also members of the adjoining university staffs.

In 1952 a new school building was completed at 707 South Wood Street, opposite the Cook County Hospital. A spacious reception room is a pleasant place to meet and visit with men from all parts of the United States, Canada and foreign countries.

Cook County Hospital has 3400 beds. It is one of the largest general hospitals in the world. It has an out-patient department which handles over 200,000 patient visits a year. This large wealth of material is available for teaching in the postgraduate school.

I chose the informal course in pediatrics because the teaching is done at the bedside of the patient and not in the lecture room. It is both comprehensive and interesting and certainly worth the time taken to study this field.

No description of Cook County Hospital is complete without a word about its resident interns. They are an interesting group of men and women from all over the world, sparked by a desire to gain the best knowledge of medicine that can be obtained. Five years of study under the staff men in Chicago and a never ending supply of difficult problems have given them a grasp of their subject which is phenomenal. Their differential diagnoses are exhaustive, logical and most stimulating.

A few highlights of the course follows:

(a) Ward rounds on the premature ward.

On entering the ward each student was required to scrub, wear a clean gown and mask. The staff man, the resident intern, the senior intern, the junior intern, the head nurse and the postgraduate student, all similarly attired, went first to see the new admissions of the past twenty-four hours. The staff man examined each child in detail, pointed out any abnormalities, discussed the immediate prenatal care and set up the routine for each child.

We then went to the ward where the prematures under two pounds were treated. Here again each child was considered and examinations believed necessary were carried out, although these are kept to an absolute minimum. The head nurse outlined the treatment and condition of the child over the past twenty-four hours. The diet was aljusted, the oxygen concentration considered, fluid balance, and the necessity for any further drugs was also discussed.

The next ward contained babies who no longer needed incubator care. Here again all abnormalities were checked by the staff man and the problems of the past twenty-four hours were discussed and a solution offered for each problem.

The final ward was the predischarge ward. From here the babies were allowed home, and a routine to be handed to the mother was also considered. The final check on the babies was made. There was ample opportunity for questions and discussions throughout the whole rounds.

(b) The outpatient clinic for rheumatic fever.

All children discharged from the children's section of the Cook County Hospital who have had rheumatic fever are brought back monthly to an out-patient clinic. This clinic is in charge of a staff and an associate. All patients were examined, throat swabs taken, and long acting penicillin was given. A summary of the child's history, his progressive lab. report and follow-up notes of his treatment since discharge from hospital were available for study and discussion.

An active social service in co-operation with Community Clubs has made summer camps available to all these children and plans were being

made to see that all children with rheumatic fever have the opportunity of going to a summer camp.

An interesting afternoon clinic of children with progressive muscular atrophy was attended with some amazement. A large number of these children were seen and greatly assisted by an interested staff man and an active physiotherapy unit. It was most gratifying to see the attention and time given to these unfortunate children.

Ward rounds in the afternoon with the resident intern was a highlight of the course. The important points in the history of each child was given, the treatment the child was receiving, consideration of the differential diagnosis and the investigations that were under way in problem cases, were most interesting. There was ample opportunity to examine each child and see the result of treatment from day to day.

Each day there was a final conference in a special amphitheatre of the hospital attended by staff men, resident internes, students from the universities and post-graduate students. Here were presented the interesting problem cases, and appropriate specialists were brought in to discuss their particular problem as it was presented.

I also had the opportunity of attending several Cook County Hospital staff conferences. These are well attended by hospital personnel and form an important nucleus in the teaching at Cook County Hospital.

My trip to Cook County Hospital in Chicago was a very pleasant and profitable experience and I would like again to thank the Manitoba Institute for the Advancement of Medical Education and Research for making available a grant that permitted me to take a course at this time.

J. E. Hudson.

Editor's Note:

Dr. J. E. Hudson of Hamiota, Manitoba is the first recipient of an award by the Manitoba Institute for the Advancement of Medical Education and Research to a general practitioner. This is an award of $400.00 to help pay the expenses to a post-graduate course. Dr. Hudson chose a course in Pediatrics at Cook County Hospital, Chicago.

Social News

Reported by K. Borthwick-Leslie, M.D.

Welcome to Doctor Leonard Bradley — newly appointed Superintendent of General Hospital. I have not had the pleasure yet of meeting Dr. Bradley personally, but have been thoroughly briefed on his capabilities via his Calgary fans.

Native to Saskatchewan (Neudorf), his medical career began as a Pediatrician, then to R.C.A.F. in 1940, and now he holds the rank of Wing Commander in the Auxiliary Force (Medical). Administrative training led to his appointment as Associate Professor of Hospital Administration at the University of Toronto, where he also became Executive Secretary of the Canadian Hospital Council and editor of its official magazine. In 1948-50 he was in charge of the studies for the Ontario health survey.

Fortunately, Dr. Bradley saw the "light" and came back home to the West, where in Calgary he has been administrator of Calgary Hospital and now fortunately again for us he has taken on the strenuous, responsible, and all important position as administrator of the General in the new expansion programme, the core of the new medical centre.

More power, and all success to you, Dr. Bradley.

♦

Congratulations to Dr. S. S. Peikoff—on being appointed official Canadian delegate to International College of Surgeons House of Delegates meeting in Chicago, September 9, 1956, at Palmerhouse Hotel. Also on being appointed as Regent for Manitoba in the International College.

♦

In July 1956, Dr. Colin C. Ferguson, Professor and Chairman, Department of Surgery, University of Manitoba, visited Medical centres in Scotland, England, Denmark and France. In Scotland, Dr. Ferguson delivered a paper to the Department of Surgery of the University of Edinburgh on "Experiences in Thoracic Surgery in Infants and Children at the Winnipeg Children's Hospital." In London, Dr. Ferguson attended the meeting of the British Association of Pediatric Surgeons, and at the meeting delivered a paper on the "Surgical Correction of Funnel Chest." In Denmark, Dr. Ferguson attended the International Congress of Pediatrics and in Paris visited cardiac surgical centres. Mrs. Ferguson accompanied him on the trip.

♦

Much activity and concentration in all Medical Circles, organizing the year's schedule of post-graduate courses, ward rounds, clinical luncheons, students' classes, and the all-important Annual Meeting of the Manitoba Medical Association—the week of October 15th.

Everyone but the stork too busy for social activities so on to the stork's report.

Dr. and Mrs. Wallace Grant very happily announce the arrival of Jennifer Rae, Sept. 7, 1956.

♦

Dr. and Mrs. Frank Boult, running true to form announce the birth of their fourth son, Brian Day, September 13, 1956. I wonder what Frank has in mind—hockey, baseball, basketball team? Surely not rugby . . . The juniors are already set for bridge, golf and curling.

♦

Dr. and Mrs. J. H. Battershill announce the birth of Peter Malcolm, August 27, 1956.

♦

Dr. and Mrs. L. S. McMorris are happy to announce the arrival of Evelyn Anne, August 25, 1956.

♦

Dr. and Mrs. Samuel Schwartz also announce a boy, Daniel Lewis, August 27, 1956.

♦

Dr. and Mrs. Colin McDonald (nee Joyce Haig) announce the arrival of Susan Evelyn, September 20, 1956.

♦

Dr. and Mrs. David Shapira announce the birth of a son, Sidney Alan, September 15, 1956, baby brother for Gary and Sheila.

♦

Dr. and Mrs. G. D. Thomson, Dauphin, Man., happily announce the birth of Rory Thomas, Sept. 18th in Dauphin.

♦

That's all. See you at the Royal Alex — Oct. 15 - 18.

The tentative programme looks most interesting, entertaining and informative.

♦

P.S. Dr. Donald Whyte, M.M.C., 1933, dropped in this p.m. for an official chat. He is supposed to be attending the meeting of the Canadian Educational Association this week, in his official capacity as Vice Chairman of the Board of Education.

Don is doing orthopedics only, in Peterboro, Ont.

The nattering parent of three—two boys and one girl. The elder ones now at the Queens U. stage. Don looks about the same, perhaps a bit more of the intellectual "brow." Nice to see old friends.

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ANNUAL MEETING

Manitoba Medical Association

(Canadian Medical Association, Manitoba Division)

Winnipeg, October 15, 16, 17, 18

Program

All Scientific and Business Sessions will be held in the Crystal Ballroom Royal Alexandra Hotel

Guest Speakers

Dr. Jacques Genest, Director Clinical Research, Hotel Dieu, Montreal, P.Q.

Dr. G. A. Hallenbeck, Surgical Section, Mayo Clinic, Rochester, Minn.

Dr. J. Renaud Lemieux, President, Canadian Medical Association, Quebec, P.Q.

Dr. Joseph Luke, Surgeon, Royal Victoria Hospital, Montreal, P.Q.

Dr. Morris Victor, Assistant Neurologist, Massachusetts General Hospital, Lecturer in Neurology, Harvard Medical School, Boston.

Sunday, October 14th

Afternoon

2.00 M.M.A. Executive Committee, Windsor Room.

Evening

6.00 Vice Regal Suite.
President's Dinner to Retiring Executive.

Monday, October 15th

Morning

9.00 Health Officers Section, Manitoba Public Health Association.

12.00 Luncheon, Health Officers Section.

Afternoon

2.00 Meeting, Health Officers Section.

2.00 Annual Business Meeting.

Evening

8.00 Annual Business Meeting —
Panel Discussion, Report of Commission re Manitoba Medical Service.
Chairman — Dr. P. L'Heureux.

Tuesday, October 16th

Morning

8.15 Registration.

8.30 Welcome by President, Manitoba Medical Association,
Dr. Ruvin Lyons

8.35 Film: Vitamin Deficiency in Pregnancy.

9.00 New Uses of Tracheotomy as an Adjunct to Surgery,
Dr. J. M. Kagan.
Dr. A. A. Klass, Chairman.

9.30 Who is Normal?
Prof. I. MacLaren Thompson.

10.00 Infections in the Newborn and their Sequelae.
Dr. E. J. S. N. Briggs.
Dr. Bruce Chown, Chairman.

10.30 Intermission — Visit the Exhibits.

11.00 The Modern Management of the Arteriosclerotic Leg,
Dr. J. C. Luke, Montreal.
Dr. John Farr, Chairman.

Noon

12.00 Luncheon, Colonial Room. Guest Speaker Dr. J. Renaud Lemieux, President, Canadian Medical Association, Quebec.

Afternoon

2.00 The Management of Arterial Hypertension,
Dr. Jacques Genest, Montreal.
Dr. R. E. Beamish, Chairman.

3.00 What'll We Give 'Em? Dr. Sydney Israels.
Dr. M. McLandress, Chairman.

3.30 Symposium on Hospital Infections,
Panel: Dr. Elinor Black, Dr. John Gemmell, Dr. L. O. Bradley, Dr. Lloyd Bartlett.
Dr. J. C. Wilt, Chairman.

Evening

6.00 Sectional Dinners.
re Manitoba Medical Service.

6.00 General Practitioners' Association of Manitoba Dinner and Meeting — Tapestry Room.

6.00 College of General Practice.
Dinner and Meeting — Tapestry Room.

6.00 Internists Section.

Wednesday, October 17th

Morning

8.15 Registration.

8.30 Film: Hypotensive Surgery.

9.00 Intra-Uterine Foetal Death after the Seventh Month.
Dr. Leon Rubin.
Dr. Elinor Black, Chairman.

9.30 The Side-Effects of the Newer Hypotensive Drugs.
Dr. Jacques Genest, Montreal.
Dr. John Gemmell, Chairman.

10.30 Intermission — Visit the Exhibits.

11.00 The Causes and Management of Leg Ulcers.
Dr. Joseph Luke, Montreal.
Dr. S. S. Peikoff, Chairman.

Noon

12.00 Luncheon, Colonial Room.
The Effect of Alcohol on the Nervous System.
Dr. Morris Victor, Boston.

Afternoon

2.00 Carcinoma of the Stomach.
Dr. G. A. Hallenback, Rochester.
Dr. M. R. MacCharles, Chairman.

3.00 Annual Business Meeting (Continued).
Discussion — Manitoba Medical Service.

Evening

8.00 Annual Business Meeting (Continued).
Presidential Address.
Dr. Ruvin Lyons.

Thursday, October 18th

Morning

8.30 Registration.

9.00 Abnormal Presentations.
Dr. R. L. Willows.
Dr. H. Guyot, Chairman.

9.30 The Increasing Problem of Drug Reaction.
Dr. A. R. Birt.
Dr. C. H. Walton, Chairman.

10.30 Intermission — Visit the Exhibits.

11.00 Some Aspects of Portal Hypertension.
Dr. G. A. Hallenbeck.
Dr. Colin Ferguson, Chairman.

Afternoon

12.00 Luncheon, Colonial Room. Guest Speaker — Hon. R. W. Bend, Minister of Health and Public Welfare, Province of Manitoba.

2.00 Cerebral Vascular Disease — Current Concepts in Diagnosis and Management.
Dr. Morris Victor.
Dr. A. Hollenberg, Chairman.

3.00 The Relationship of Disordered Fat Metabolism to Coronary Athero Sclerosis.
Dr. P. T. Green.
Dr. W. F. Perry, Chairman.

3.30 The Management of Metastatic Breast Cancer.
Dr. R. L. Cooke.
Dr. K. R. Trueman, Chairman.

Evening

6.30 Reception — Tea Lounge.
7.15 Dinner — Alexandra Room.
9.00 Dance — Crystal Ballroom.

General Practitioners' Annual Dinner

The Annual Dinner of the General Practitioners Associations of Manitoba, will be held at the Royal Alexandra Hotel, Winnipeg on October 16th, 1956. The same to be followed by the Annual meeting of the G.P.A.M. and the Annual meeting of the Manitoba section of the College of General Practice.

Ladies' Programme

Under the Chairmanship of Mrs. Ruvin Lyons, the Ladies' Committee is presenting a programme that will be of interest to all ladies attending. Full details of the programme of social events will be announced as arrangements are completed. Registration Booth, Banquet Hall, Royal Alexandra Hotel.

Scientific Exhibits — — — — Commercial Exhibits

Association Page

Reported by M. T. Macfarland, M.D.

The Canadian Medical Association

Toronto 5, Ontario

August 3, 1956.

To the Secretaries of The Divisions

Dear Doctor Macfarland:

You have doubtless heard through the news media of the very gratifying development in the removal of the geographic limitation on convention expenses by self-employed taxpayers.

Hansard of Tuesday, July 31st, indicates that the Minister of National Revenue moved, and the Minister of Finance accepted, with the hearty approval of the opposition, an amendment deleting the words "in Canada" on the convention amendment to the Income Tax Act.

Announcement of this very successful conclusion of a long campaign by the Income Tax Committee will be made in the August 15th issue of the C.M.A.J. in the attached brief article. You are at liberty to notify your members of this change and I am sure that they will be pleased.

In letters of appreciation to the Ministers of Finance and National Revenue I have renewed the offer of the Income Tax Committee to assist the Department of National Revenue in framing and applying rules designed to prevent abuses of the new privilege of international travel. I am as yet not informed of any limitation which may pertain aside from the statutory one of two meetings per year which apply to the "business or profession" of the taxpayer. It would appear that "conventions" may be defined as actual meetings of organized bodies and not postgraduate courses. It would appear reasonable that membership in the organization would be a requirement or that the doctor be an invited guest speaker or official delegate. Certificates of attendance to substantiate a claim for personal expenses is very likely to be a continuing requirement. This amendment to the Act has no application to returns by salaried taxpayers.

From my contacts with the Department of National Revenue I gather that every effort will be made to protect the treasury from claims which they would classify as joy rides. We must agree that the new privilege should not be jeopardized by frivolous claims and excessive demands and I would appreciate the advice of my fellow secretaries on the principles which we should put forward to prevent possible abuses.

Yours faithfully,

A. D. Kelly,
General Secretary.

Convention Expenses Deductible

Through the columns of the Journal the profession has been kept informed of the efforts of the Income Tax Committee to retrieve the situation presented by the adverse rulings of the Income Tax Appeal Board and the Exchequer Court of Canada, in the matter of deductibility of the expenses of attending medical meetings.

In the issue of February 1st we published the submission of the Income Tax Committee to the Honourable Minister of Finance on this and other matters, and indicated that copies of this brief had been sent to all members of Parliament. In the March 1st issue there was reproduced the annual memorandum on income tax returns by members of the medical profession. The uncertainties of the situation at that time dictated a very cautious statement under the heading "Convention Expenses."

In the issue of April 1st reference was made to the Budget Speech of a few days previously in which the Minister of Finance had announced his intention to amend the Income Tax Act to provide for the deductibility of the expenses of two conventions annually in Canada. While deriving considerable satisfaction at the admission of the principle, it was the view of your Committee that the geographic limitation was unfortunate. Representations were promptly and energetically made at this time and when the amending act was debated on first reading in Parliament. We urged that additional or alternative meetings in the United States or further afield should be recognized in view of the international characteristics of medicine and the commitments of many Canadian doctors as members of international societies. Little encouragement was given to your negotiators that any geographic relaxation would be granted and the Committee was obligated to conclude that the extension to cover meetings outside of Canada remained to be fought for another day.

The last act of the drama was played on July 31st in the House of Commons when the Minister of National Revenue proposed, and the Minister of Finance accepted, a modification of the amendment to the Income Tax Act, deleting the words "in Canada." A spokesman for the opposition expressed hearty approval of this change and indicated that a similar amendment had been ready for presentation. The removal of this geographic limitation has the effect of providing in the Income Tax Act itself authority for self-employed taxpayers to claim as expenses of their business or profession the cost of two conventions annually wherever they may be held.

Members of the medical profession and their patients are the chief beneficiaries of this enlightened piece of legislation because doctors attend meetings to teach and to learn. Members of The Association will be grateful to their Income Tax Committee for having waged so successful a campaign and to the members of Parliament of all parties for their acceptance of a fair and reasonable recommendation.

Department of Health and Public Welfare

Comparisons Communicable Diseases — Manitoba (Whites and Indians)

DISEASES	1956 June 17 to July 14,'56	1956 May 20 to June 16,'56	1955 June 19 to July 16,'55	1955 May 22 to June 18,'55	Total Jan. 1 to July 14,'56	Total Jan. 1 to July 16,'55
Anterior Poliomyelitis	0	1	3	2	5	8
Chickenpox	70	149	46	93	647	854
Diphtheria	0	2	0	0	2	1
Diarrhoea and Enteritis, under 1 year	8	10	11	15	63	59
Diphtheria Carriers	0	2	0	0	2	2
Dysentery—Amoebic	0	0	0	0	0	0
Dysentery—Bacillary	0	1	4	1	9	9
Erysipelas	0	1	0	1	11	8
Encephalitis	0	0	0	0	0	0
Influenza	2	15	13	5	73	182
Measles	84	127	67	117	1048	2005
Measles—German	6	15	4	1	153	60
Meningococcal Meningitis	1	1	1	0	4	12
Mumps	40	100	56	66	909	862
Ophthalmia Neonatorum	0	0	0	0	0	1
Psittacosis	0	1	0	0	1	0
Puerperal Fever	0	0	0	1	1	1
Scarlet Fever	6	3	14	14	92	122
Septic Sore Throat	0	0	2	3	4	15
Smallpox	0	0	0	0	0	0
Tetanus	0	2	0	0	2	0
Trachoma	0	0	0	0	0	0
Tuberculosis	40	43	43	103	267	385
Typhoid Fever	0	0	0	0	0	1
Typhoid Paratyphoid	0	0	0	0	1	0
Typhoid Carriers	0	0	0	0	0	0
Undulant Fever	1	1	1	1	7	6
Whooping Cough	33	47	55	57	229	455
Gonorrhoea	70	86	109	76	682	571
Syphilis	6	5	7	6	39	62
Jaundice Infectious	8	15	22	27	168	195
Tularemia	0	0	1	0	0	3

Four-Week Period June 17th to July 14th, 1956

DISEASES (White Cases Only) *Approximate population.	*849,000 Manitoba	*861,000 Saskatchewan	*2,825,000 Ontario	*2,952,000 Minnesota
Anterior Poliomyelitis	—	2	5	6
Chickenpox	70	1	817	—
Diarrhoea & Enteritis, under 1 yr.	8	12	—	—
Diphtheria	—	—	—	—
Diphtheria Carriers	—	1	—	—
Dysentery—Amoebic	—	—	—	—
Bacillary	—	14	4	4
Encephalitis Infectious	—	2	—	—
Erysipelas	—	—	—	—
Influenza	2	—	3	4
Jaundice Infectious	8	87	15	31
Measles	84		1714	95
German Measles	6	1	875	—
Meningitis Meningococcus	1	2	3	—
Mumps	40	1	631	—
Ophthal. Neonat.	—	—	—	—
Puerperal Fever	—	—	—	—
Scarlet Fever	6	6	219	20
Septic Sore Throat	—	24	3	29
Smallpox	—	—	—	—
Tetanus	—	—	—	—
Trachoma	—	—	—	—
Tuberculosis	40	27	82	75
Tularemia	—	—	—	1
Typhoid Fever	—	2	5	1
Typh. Para-typhoid	—	—	—	—
Typhoid Carriers	—	—	—	—
Undulant Fever	1	—	1	5
Whooping Cough	33	15	49	1
Gonorrhoea	70	—	118	—
Syphilis	6	—	21	—
Salmonellosis	—	1	—	—

DEATHS FROM REPORTABLE DISEASES

July, 1956

Urban—Cancer, 63; Pneumonia, Lobar (490), 4; Pneumonia (other forms), 12; Syphilis, 1; Tuberculosis, 4; Diarrhoea and Enteritis, 3; Septicaemia and Pyaemia, 1. Other deaths under 1 year, 20. Other deaths over 1 year, 193. Stillbirths, 17. Total, 318.

Rural—Cancer, 30; Influenza, 1; Measles, 1; Pneumonia, Lobar (490), 1; Pneumonia (other forms), 5; Syphilis, 1; Tuberculosis, 2; Whooping Cough, 1. Other deaths under 1 year, 22. Other deaths over 1 year, 180. Stillbirths, 11. Total, 255.

Indians — Pneumonia, Lobar (490), 2; Pneumonia (other forms), 3; Tuberculosis, 2; Diarrhoea and Enteritis, 1. Other deaths under 1 year, 2. Other deaths over 1 year, 3. Stillbirths, 1. Total, 14.

◆

Poliomyelitis—The month of July is over with at the time of writing (August 7) and no cases reported.

Jaundice Infectious—Still scattered throughout the province.

All communicable diseases showed a lower incidence last month.

Detailmen's Directory

Representing Review Advertisers in this issue, whose names are not listed under a business address.

Ayerst, McKenna and Harrison
W. R. Card — 40-7115
C. G. Savage — SU3-4558
Jack R. Ostrow — ED 4-3240

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